10/823,819

```
=> fil JICST-EPLUS, CABA, BIOSIS, LIFESCI, CONFSCI, DISSABS, SCISEARCH
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=> d que l137

L122         254 SEA FLEISZIG S?/AU
L123       16140 SEA EVANS D?/AU
L124        1188 SEA SACK R?/AU
L134        1201 SEA COLLECTIN OR COLLECTINS OR COLLAGEN LIKE LECTIN#
L135        3121 SEA (SP OR SURFACTANT(1W) PROTEIN)(W) D
L137           8 SEA (L122 AND L123 AND L124) OR ((L122 OR L123 OR L124) AND
                 (L134 OR L135))


=> fil drugu; d que l108; d que l118; d que l111

FILE 'DRUGU' ENTERED AT 14:55:26 ON 04 OCT 2006
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FILE LAST UPDATED: 4 OCT 2006       <20061004/UP>
>>>  DERWENT DRUG FILE (SUBSCRIBER)  <<<

>>>  FILE COVERS 1983 TO DATE  <<<
>>>  THESAURUS AVAILABLE IN /CT  <<<


L108           0 SEA FILE=DRUGU ABB=ON  FLEISZIG S?/AU



L109         397 SEA FILE=DRUGU ABB=ON  EVANS D?/AU
L110          31 SEA FILE=DRUGU ABB=ON  SACK R?/AU
L115           7 SEA FILE=DRUGU ABB=ON  COLLECTIN OR COLLECTINS OR COLLAGEN
                  LIKE LECTIN#
L116          94 SEA FILE=DRUGU ABB=ON  (SP OR SURFACTANT(1W)PROTEIN)(W)D
L118           0 SEA FILE=DRUGU ABB=ON  (L109 OR L110) AND (L115 OR L116)
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```
L109          397 SEA FILE=DRUGU ABB=ON  EVANS D?/AU
L110           31 SEA FILE=DRUGU ABB=ON  SACK R?/AU
L111            0 SEA FILE=DRUGU ABB=ON  L109 AND L110


=> fil medl; d que l42; fil embase; d que l66; fil wpix; d que l92

FILE 'MEDLINE' ENTERED AT 14:55:28 ON 04 OCT 2006

 FILE LAST UPDATED: 3 Oct 2006 (20061003/UP).  FILE COVERS 1950 TO DATE.

 On December 11, 2005, the 2006 MeSH terms were loaded.

 The MEDLINE reload for 2006 is now (26 Feb.) available.  For details
 on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).
 See also:

    http://www.nlm.nih.gov/mesh/
    http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

 OLDMEDLINE is covered back to 1950.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2006 vocabulary.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


L33            48 SEA FILE=MEDLINE ABB=ON  FLEISZIG S?/AU
L34          4088 SEA FILE=MEDLINE ABB=ON  EVANS D?/AU
L35           413 SEA FILE=MEDLINE ABB=ON  SACK R?/AU
L36          2293 SEA FILE=MEDLINE ABB=ON  COLLECTINS+NT/CT
L42             1 SEA FILE=MEDLINE ABB=ON  (L33 AND L34 AND L35) OR ((L33 OR L34
                  OR L35) AND L36)



FILE 'EMBASE' ENTERED AT 14:55:28 ON 04 OCT 2006
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 FILE COVERS 1974 TO 4 Oct 2006 (20061004/ED)

 EMBASE has been reloaded. Enter HELP RLOAD for details.

 EMBASE is now updated daily.  SDI frequency remains weekly (default)
 and biweekly.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


L56            44 SEA FILE=EMBASE ABB=ON  FLEISZIG S?/AU
L57          3264 SEA FILE=EMBASE ABB=ON  EVANS D?/AU
L58           299 SEA FILE=EMBASE ABB=ON  SACK R?/AU
L59           221 SEA FILE=EMBASE ABB=ON  COLLECTIN/CT
L60           535 SEA FILE=EMBASE ABB=ON  SURFACTANT PROTEIN D/CT
L61           375 SEA FILE=EMBASE ABB=ON  SURFACTANT ASSOCIATED PROTEIN/CT
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```
L66             1 SEA FILE=EMBASE ABB=ON  (L56 AND L57 AND L58) OR ((L56 OR L57
                  OR L58) AND (L59 OR L60 OR L61))


FILE 'WPIX' ENTERED AT 14:55:28 ON 04 OCT 2006
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FILE LAST UPDATED:          2 OCT 2006      <20061002/UP>
MOST RECENT DERWENT UPDATE:     200663        <200663/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

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    PLEASE VISIT:
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http://www.stn-international.de/stndatabases/details/ipc_reform.html and
http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf <<<

>>> FOR FURTHER DETAILS ON THE FORTHCOMING DERWENT WORLD PATENTS
    INDEX ENHANCEMENTS PLEASE VISIT:
http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<
'BI ABEX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE


L82             3 SEA FILE=WPIX ABB=ON  FLEISZIG S?/AU
L83           848 SEA FILE=WPIX ABB=ON  EVANS D?/AU
L84            32 SEA FILE=WPIX ABB=ON  SACK R?/AU
L85            51 SEA FILE=WPIX ABB=ON  COLLECTIN/BI,ABEX OR COLLECTINS/BI,ABEX
                  OR COLLAGEN LIKE LECTIN#/BI,ABEX
L86           318 SEA FILE=WPIX ABB=ON  SPD/BI,ABEX OR (SP/BI,ABEX OR SURFACTANT/
                  BI,ABEX(1W)PROTEIN/BI,ABEX)(W)D/BI,ABEX
L92             1 SEA FILE=WPIX ABB=ON  (L82 AND L83 AND L84) OR ((L82 OR L83 OR
                  L84) AND (L85 OR L86))


=> fil capl; d que l1; d que l12; d que l5

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FILE COVERS 1907 - 4 Oct 2006  VOL 145 ISS 15
FILE LAST UPDATED: 3 Oct 2006  (20061003/ED)
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L1             1 SEA FILE=CAPLUS ABB=ON  US2004-823819/AP



L2            33 SEA FILE=CAPLUS ABB=ON  FLEISZIG S?/AU
L3          5041 SEA FILE=CAPLUS ABB=ON  EVANS D?/AU
L4           246 SEA FILE=CAPLUS ABB=ON  SACK R?/AU
L6          9103 SEA FILE=CAPLUS ABB=ON  COLLECTIN#/OBI
L7          4798 SEA FILE=CAPLUS ABB=ON  SURFACTANT/OBI(L)PROTEIN#/OBI
L12            3 SEA FILE=CAPLUS ABB=ON  (L2 OR L3 OR L4) AND (L6 OR L7)



L2            33 SEA FILE=CAPLUS ABB=ON  FLEISZIG S?/AU
L3          5041 SEA FILE=CAPLUS ABB=ON  EVANS D?/AU
L4           246 SEA FILE=CAPLUS ABB=ON  SACK R?/AU
L5             2 SEA FILE=CAPLUS ABB=ON  L2 AND L3 AND L4


=> s l1,l5,l12

L142           3 (L1 OR L5 OR L12)

=> dup rem l42,l142,l92,l66,l137
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PROCESSING COMPLETED FOR L42
PROCESSING COMPLETED FOR L142
PROCESSING COMPLETED FOR L92
PROCESSING COMPLETED FOR L66
PROCESSING COMPLETED FOR L137
L143           8 DUP REM L42 L142 L92 L66 L137 (6 DUPLICATES REMOVED)
                 ANSWER '1' FROM FILE MEDLINE
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ANSWERS '2-3' FROM FILE CAPLUS
ANSWER '4' FROM FILE WPIX
ANSWERS '5-7' FROM FILE BIOSIS
ANSWER '8' FROM FILE SCISEARCH

=> d ibib ed abs 1-8

L143 ANSWER 1 OF 8 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2005151833 MEDLINE Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PubMed ID: 15784557
TITLE: Surfactant protein D is present in human tear fluid and the cornea and inhibits epithelial cell invasion by Pseudomonas aeruginosa.
AUTHOR: Ni Minjian; **Evans David J**; Hawgood Samuel; Anders E Margot; **Sack Robert A**; **Fleiszig Suzanne M J**
CORPORATE SOURCE: School of Optometry, University of California, Berkeley, CA 94720-2020.. fleiszig@socrates.berkeley.edu
CONTRACT NUMBER: R01-EY11221 (NEI)
SOURCE: Infection and immunity, (2005 Apr) Vol. 73, No. 4, pp. 2147-56.
Journal code: 0246127. ISSN: 0019-9567.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 24 Mar 2005
Last Updated on STN: 15 Apr 2005
Entered Medline: 14 Apr 2005

ED Entered STN: 24 Mar 2005
Last Updated on STN: 15 Apr 2005
Entered Medline: 14 Apr 2005

AB We have previously shown that human tear fluid protects corneal epithelial cells against Pseudomonas aeruginosa in vitro and in vivo and that protection does not depend upon tear bacteriostatic activity. We sought to identify the responsible tear component(s). The hypothesis tested was that collectins (collagenous calcium-dependent lectins) were involved. Reflex tear fluid was collected from healthy human subjects and examined for collectin content by enzyme-linked immunosorbent assay (ELISA) and Western blot with antibody against surfactant protein D (SP-D), SP-A, or mannose-binding lectin (MBL). SP-D, but not SP-A or MBL, was detected by ELISA of human reflex tear fluid. Western blot analysis of whole tears and of high-performance liquid chromatography tear fractions confirmed the presence of SP-D, most of which eluted in the same fraction as immunoglobulin A. SP-D tear concentrations were calculated at approximately 2 to 5 microg/ml. Depletion of SP-D with mannan-conjugated Sepharose or anti-SP-D antibody reduced the protective effect of tears against P. aeruginosa invasion. Recombinant human or mouse SP-D used alone reduced P. aeruginosa invasion of epithelial cells without detectable bacteriostatic activity or bacterial aggregation. Immunofluorescence microscopy revealed SP-D antibody labeling throughout the corneal epithelium of normal, but not gene-targeted SP-D knockout mice. SP-D was also detected in vitro in cultured human and mouse corneal epithelial cells. In conclusion, SP-D is present in human tear fluid and in human and mouse corneal epithelia. SP-D is involved in human tear fluid protection against P. aeruginosa invasion. Whether SP-D plays other roles in the regulation of other innate or adaptive immune responses at the ocular surface, as it does in the airways, remains to be explored.

L143 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:995762 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 141:388776
TITLE: Methods and compositions with collectins for treating ocular disease
INVENTOR(S): **Fleiszig, Suzanne M. j.; Evans, David J.; Sack, Robert A.**
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004229802	A1	20041118	US 2004-823819	20040414 <--
WO 2004091436	A2	20041028	WO 2004-US11474	20040414
WO 2004091436	A3	20050224		

W: AE, AE, AG, AL, AL, AM, AM, AM, AT, AT, AU, AZ, AZ, BA, BB, BG, BG, BR, BR, BW, BY, BY, BZ, BZ, CA, CH, CN, CN, CO, CO, CR, CR, CU, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EC, EE, EE, EG, EG, ES, ES, FI, FI, GB, GD, GE, GE, GH, GM, HR, HR, HU, HU, ID, IL, IN, IS, JP, JP, KE, KE, KG, KG, KP, KP, KP, KR, KR, KZ, KZ, KZ, LC, LK, LR, LS, LS, LT, LU, LV, MA, MD, MD, MG, MK, MN, MW, MX, MX, MZ, MZ, NA
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-462913P P 20030415
ED Entered STN: 19 Nov 2004
AB The use of collectins and/or surfactant proteins for the treatment and prevention of ocular disease. Surfactant protein-D (SP-D) was found in large amts. in human tears. SP-D protected corneal cells against invasion by Pseudomonas aeruginosa.

L143 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1959:65602 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 53:65602
ORIGINAL REFERENCE NO.: 53:11901g
TITLE: Useful fraction collector for small-scale vacuum distillations
AUTHOR(S): **Evans, D. W. S.**
CORPORATE SOURCE: Univ. Nottingham, UK
SOURCE: Chemistry & Industry (London, United Kingdom) (1959) 219-20
CODEN: CHINAG; ISSN: 0009-3068
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
ED Entered STN: 22 Apr 2001
AB A modified Kon rectangle glass apparatus for the separation of small amts. (2-3 ml.) of vacuum distillate is illustrated.

L143 ANSWER 4 OF 8 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2004-795179 [78] WPIX
DOC. NO. NON-CPI: N2004-626742
DOC. NO. CPI: C2004-277511
TITLE: Treating ocular disease such as dry eye, keratitis and conjunctivitis comprises administering a surfactant protein (especially **collectin**) to the eye.
DERWENT CLASS: B04 D22 P32
INVENTOR(S): **EVANS, D J; FLEISZIG, S M J; SACK, R A; FLEISZIG, S; SACK, R**
PATENT ASSIGNEE(S): (EVAN-I) EVANS D J; (FLEI-I) FLEISZIG S M J; (SACK-I) SACK R A; (REGC) UNIV CALIFORNIA; (UYNY) UNIV NEW YORK STATE RES FOUND
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004091436	A2	20041028	(200478)*	EN	52

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

US 2004229802 A1 20041118 (200478)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004091436	A2	WO 2004-US11474	20040414
US 2004229802	A1 Provisional	US 2003-462913P	20030415
		US 2004-823819	20040414

PRIORITY APPLN. INFO: US 2003-462913P 20030415; US 2004-823819 20040414

ED 20041206
AN 2004-795179 [78] WPIX
AB WO2004091436 A UPAB: 20041206
NOVELTY - Treatment of an ocular disease in subject involves administering a pharmaceutical composition comprising a surfactant protein (especially **collectin**) into the eye of a subject.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:
(1) an ophthalmic composition comprising a surfactant protein (**collectin**) and a liquid aqueous medium compatible with application to a mammalian eye;
(2) kit for the treatment of a subject having an ocular disease comprising a pharmaceutical composition containing a **collectin** and instructions for the administration of the **collectin**; and (3) an antimicrobial lens (preferably a contact lens) comprising surfactant protein (**collectin**).
ACTIVITY - Ophthalmological; Antiinflammatory.
MECHANISM OF ACTION - None given.
USE - As an artificial tear composition for storing, cleaning, re-writing or disinfecting a contact lens; for treating or preventing ocular diseases e.g. dry eye, keratitis, caused by microbe such as bacterial, viral, fungal or protozoan pathogen, gram-negative bacterium such as Pseudomonas aeruginosa in a contact-lens wearer (claimed).

ADVANTAGE - The treatment protects eye from ocular disease. The **surfactant protein-D** is found in large amounts in human tears and protects corneal cells against microbial invasion e.g. Pseudomonas aeruginosa invasion.
Dwg.0/7

L143 ANSWER 5 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 2
ACCESSION NUMBER: 2005:457944 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV200510252412
TITLE: **Surfactant Protein D** is present in tear fluid and corneal epithelium and inhibits corneal epithelial cell invasion by Pseudomonas aeruginosa.
AUTHOR(S): Ni, M. [Reprint Author]; **Evans, D. J.**; **Sack, R. A.**; Anders, M.; Hawgood, S.; **Fleiszig, S. M. J.**
CORPORATE SOURCE: Univ Calif Berkeley, Sch Optometry, Berkeley, CA 94720 USA
SOURCE: IOVS, (APR 2004) Vol. 45, No. Suppl. 2, pp. U486.
Meeting Info.: Annual Meeting of the Association-for-Research-in-Vision-and-Ophthalmology. Ft Lauderdale, FL, USA. April 24 -29, 2004. Assoc Res Vis & Ophthalmol.
CODEN: IOVSDA. ISSN: 0146-0404.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Nov 2005
Last Updated on STN: 9 Nov 2005
ED Entered STN: 9 Nov 2005
Last Updated on STN: 9 Nov 2005
AB Purpose: We have previously shown that human tear fluid protected corneal epithelial cells against P. aeruginosa (PA) in vitro and in vivo, and that protection did not depend upon tear bacteriostatic activity. We sought to identify the responsible tear component(s). The hypothesis tested was that **collectins** (collagenous, calcium-dependent lectins) were involved, based upon recent findings of their activities in human airway secretions.Methods: Reflex tear fluid was collected from healthy human subjects and examined for **collectin** content by ELISA and Western blot using antibody against **surfactant protein D (SP-D)**, SP-A or mannose binding lectin (MBL). To test the role of detected **SP-D** in protection against PA invasion of corneal epithelial cells it was subtracted from human tear fluid using two separate methods; mannan-conjugated sepharose and anti-**SP-D** antibody. **SP-D** subtracted tear fluid was compared to whole team for protective effects against PA invasion using gentamicin survival assays. In other experiments, recombinant human and mouse **SP- D** were compared to buffer-only controls for their effect. on PA invasion. **SP-D** distribution in the corneal epithelium of C57BL/6 mice was examined by immunofluorescence with gene-targeted C57BL/6 "knockout" mice (deficient in **SP-D** gene expression) used as negative controls.Results: **SP-D**, but not SNA or MBL, was detected by ELISA of human reflex tear fluid. Western blot analysis of whole team and of HPLC fractions confirmed thepresence of **SP-D**, most of which eluted in the same fraction as IgA. **SP-D** tearconcentrations ranged train 5 to 70 mu g/ml. Removal of **SP-D** from human tear fluid reduced its protective activity against PA invasion ($p = 0.03$, mannan binding method; $p = 0.04$, antibody method). Both human ($p = 0.03$) and mons, ($p = 0.01$) recombinant **SP-D** reduced PA invasion, without detectable bacteriostaticactivity. Immunoflumesence revealed **SP-D** antibody labeling throughout the corneal epithelium of normal, but not **SP-D** knockout, to ice.Conclusions: We have presented data that a mutant strain of PA defective in the early stages of biofilm formation Inks virulence in a murine model of corneal infection. This suggests that in vitro models of biofilm formation may be useful in identifying novel genes involved in the ocular virulence of PA and supports

the hypothesis that there is a relationship between biofilm formation and corneal infection.

L143 ANSWER 6 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:43563 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV200600052764
TITLE: Pseudomonas aeruginosa exposure regulates **surfactant protein D** production by human corneal epithelial cells.
AUTHOR(S): Ni, M. [Reprint Author]; **Evans, D. J.**; **Fleiszig, S. M. J.**
SOURCE: IOVS, (2005) Vol. 46, No. Suppl. S, pp. 896. Meeting Info.: Annual Meeting of the Association-for-Research-in-Vision-and-Ophthalmology. Ft Lauderdale, FL, USA. May 01 -05, 2005. Assoc Res Vis & Ophthalmol. CODEN: IOVSDA. ISSN: 0146-0404.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Jan 2006
Last Updated on STN: 4 Jan 2006
ED Entered STN: 4 Jan 2006
Last Updated on STN: 4 Jan 2006
AB Purpose: We previously showed that **SP-D** was present in human tear fluid and that it protected corneal epithelial cells against Pseudomonas aeruginosa invasion in vitro. In this study, we explored the expression of **SP-D** in corneal epithelium and then examined the effect of P. aeruginosa exposure. Methods: Primary cultures of mouse corneal epithelial cells were prepared from female 8-12 week old wild type C57BL/6 mice and gene-targeted **SP-D** deficient mice. SDS-PAGE and Western blot were performed to detect the **SP-D** level in cultured corneal epithelial cell lysates or cell growth media. To determine whether P. aeruginosa exposure can regulate **SP-D** expression, SV 40-immortalized human corneal epithelial cells were stimulated with 2 x 107 or 2 x 1010 heat killed bacteria (invasive strain PAK) or were sham inoculated. After stimulation, cells were lysed and **SP-D** quantified using Western Blot. The effect of bacterial lipopolysaccharide (LPS) on **SP-D** expression was explored using two different mutants withdefects in LPS core and O antigen. A fliC mutant was used to examine the roleof bacterial flagellin. Results: **SP-D** was detected in primary cultured mouse corneal epithelial cells derived from C57BL/6 mice, but not in lysates of corneal epithelial cells derived from **SP-D** deficient mice. **SP-D** was also detected extracellularly in cultured corneal epithelial cell growth media. Cells treated with heat killed wild type P. aeruginosa showed a strong dose-dependent upregulation of **SP-D** production in both cell lysates and cellular secretions. LPS and flagellin mutants, however, were each defective in their ability to upregulate **SP-D** in cell lysates and cell secretions. Conclusions: Corneal epithelialcells were found to make and secrete **SP-D** and as such could contribute to tear fluid **SP-D** level. **SP- D** expression in human corneal epithelial cells was strongly upregulated when cells were exposed to P. aeruginosa, which involved bacterial LPS and flagellin. These results suggest that **SP- D** is an inducible factor involved in innate immunity against P. aeruginosa invasion, and that induction could involve TLR signaling.

L143 ANSWER 7 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1970:55039 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV197006055039; BR06:55039
TITLE: AN APPROACH TO THE SYNTHESIS OF THE HASUBANAN CARBOCYCLIC SYSTEM.

AUTHOR(S): **EVANS D A**
SOURCE: Tetrahedron Letters, (1969) Vol. 20, pp. 1573-1576.
CODEN: TELEAY. ISSN: 0040-4039.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: Unavailable

L143 ANSWER 8 OF 8 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:636181 SCISEARCH Full-text<<LOGINID::20061004>>
THE GENUINE ARTICLE: 911CZ
TITLE: Pseudomonas aeruginosa exposure regulates **surfactant protein D** production by human corneal epithelial cells
AUTHOR: Ni M (Reprint); **Evans D J; Fleiszig S M J**
SOURCE: INVESTIGATIVE OPHTHALMOLOGY & VISUAL SCIENCE, (2005) Vol. 46, Supp. [S], pp. U163-U163. MA 896.
ISSN: 0146-0404.
PUBLISHER: ASSOC RESEARCH VISION OPHTHALMOLOGY INC, 12300 TWINBROOK PARKWAY, ROCKVILLE, MD 20852-1606 USA.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
ENTRY DATE: Entered STN: 29 Jun 2005
Last Updated on STN: 15 Dec 2005
ED Entered STN: 29 Jun 2005
Last Updated on STN: 15 Dec 2005

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=> => fil JICST-EPLUS, CABA, BIOSIS, LIFESCI, CONFSCI, DISSABS, SCISEARCH
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=> d que l141

L125     519542 SEA EYE?
L126     183964 SEA OCULAR? OR INTRAOCULAR?
L127     149128 SEA OPHTHALM?
L128     150518 SEA LENS OR LENSES
L129      16862 SEA KERATITIS
L130      38386 SEA TEAR#
L132       1648 SEA XEROPHTHALM?
L133      17416 SEA SJOGREN?
L134       1201 SEA COLLECTIN OR COLLECTINS OR COLLAGEN LIKE LECTIN#
L135       3121 SEA (SP OR SURFACTANT(1W) PROTEIN)(W) D
L136        177 SEA CONJUNCTIVITIS SICCA
L138         34 SEA (L134 OR L135) AND (L125 OR L126 OR L127 OR L128 OR L129
                OR L130 OR L132 OR L133 OR L136)
L140         64 SEA L134(W) RECEPTOR#
L141         31 SEA L138 NOT L140


=> fil drugu; d que l121

FILE 'DRUGU' ENTERED AT 14:58:33 ON 04 OCT 2006
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FILE LAST UPDATED: 4 OCT 2006       <20061004/UP>
>>> DERWENT DRUG FILE (SUBSCRIBER)  <<<

>>> FILE COVERS 1983 TO DATE  <<<
>>> THESAURUS AVAILABLE IN /CT  <<<



L112       7295 SEA FILE=DRUGU ABB=ON  EYE+NT/CT
L113      14910 SEA FILE=DRUGU ABB=ON  EYE -DISEASE/CT OR EYE-DISEASE+NT/CT
L115          7 SEA FILE=DRUGU ABB=ON  COLLECTIN OR COLLECTINS OR COLLAGEN
```

```
                LIKE LECTIN#
L116          94 SEA FILE=DRUGU ABB=ON  (SP OR SURFACTANT(1W)PROTEIN)(W)D
L119         983 SEA FILE=DRUGU ABB=ON  TEAR#
L120        1525 SEA FILE=DRUGU ABB=ON  LENS OR LENSES
L121           0 SEA FILE=DRUGU ABB=ON  (L115 OR L116) AND (L119 OR L120 OR
                L112 OR L113)


=> fil capl; d que l16; d que l18; d que l19; d que l32

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FILE COVERS 1907 - 4 Oct 2006  VOL 145 ISS 15
FILE LAST UPDATED: 3 Oct 2006  (20061003/ED)

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'OBI' IS DEFAULT SEARCH FIELD FOR 'CAPLUS' FILE


L6          9103 SEA FILE=CAPLUS ABB=ON  COLLECTIN#/OBI
L8         26855 SEA FILE=CAPLUS ABB=ON  EYE#/OBI(L)(DISEASE#/OBI OR DISORDER#/O
                 BI)
L9          8564 SEA FILE=CAPLUS ABB=ON  OCULAR/OBI
L10        10251 SEA FILE=CAPLUS ABB=ON  OPHTHALM?/OBI
L11         4763 SEA FILE=CAPLUS ABB=ON  CONTACT LENS?/OBI
L15          225 SEA FILE=CAPLUS ABB=ON  L6 NOT COLLECTING/OBI
L16            5 SEA FILE=CAPLUS ABB=ON  L15 AND (L8 OR L9 OR L10 OR L11)



L6          9103 SEA FILE=CAPLUS ABB=ON  COLLECTIN#/OBI
L15          225 SEA FILE=CAPLUS ABB=ON  L6 NOT COLLECTING/OBI
L17          171 SEA FILE=CAPLUS ABB=ON  ANTIMICROBIAL#/OBI(L)LENS?/OBI
L18            1 SEA FILE=CAPLUS ABB=ON  L17 AND L15



L7          4798 SEA FILE=CAPLUS ABB=ON  SURFACTANT/OBI(L)PROTEIN#/OBI
L13          635 SEA FILE=CAPLUS ABB=ON  SP D/OBI
L17          171 SEA FILE=CAPLUS ABB=ON  ANTIMICROBIAL#/OBI(L)LENS?/OBI
L19            1 SEA FILE=CAPLUS ABB=ON  (L7 OR L13) AND L17
```

```
L7          4798 SEA FILE=CAPLUS ABB=ON  SURFACTANT/OBI(L)PROTEIN#/OBI
L8         26855 SEA FILE=CAPLUS ABB=ON  EYE#/OBI(L)(DISEASE#/OBI OR DISORDER#/O
                 BI)
L9          8564 SEA FILE=CAPLUS ABB=ON  OCULAR/OBI
L10        10251 SEA FILE=CAPLUS ABB=ON  OPHTHALM?/OBI
L11         4763 SEA FILE=CAPLUS ABB=ON  CONTACT LENS?/OBI
L13          635 SEA FILE=CAPLUS ABB=ON  SP D/OBI
L24        76046 SEA FILE=CAPLUS ABB=ON  EYE/CT
L25         2234 SEA FILE=CAPLUS ABB=ON  TEAR#/CW
L29          327 SEA FILE=CAPLUS ABB=ON  (L7 OR L13)(L)(THU OR PAC OR PKT OR
                 DMA)/RL
L31          419 SEA FILE=CAPLUS ABB=ON  SURFACTANT FREE/OBI
L32            8 SEA FILE=CAPLUS ABB=ON  L29 AND (L8 OR L9 OR L10 OR L11 OR L24
                 OR L25) NOT L31


=> s l16,l18,l19,l32 not l142

L144          11 (L16 OR L18 OR L19 OR L32) NOT L142

=> fil embase; d que l73; d que l76; d que l77; d que l81

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 FILE COVERS 1974 TO 4 Oct 2006 (20061004/ED)

 EMBASE has been reloaded. Enter HELP RLOAD for details.

 EMBASE is now updated daily.  SDI frequency remains weekly (default)
 and biweekly.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


L59          221 SEA FILE=EMBASE ABB=ON  COLLECTIN/CT
L60          535 SEA FILE=EMBASE ABB=ON  SURFACTANT PROTEIN D/CT
L61          375 SEA FILE=EMBASE ABB=ON  SURFACTANT ASSOCIATED PROTEIN/CT
L62       302195 SEA FILE=EMBASE ABB=ON  EYE DISEASE+NT/CT
L72          665 SEA FILE=EMBASE ABB=ON  (L59 OR L60 OR L61)(L)EC/CT
L73            1 SEA FILE=EMBASE ABB=ON  (L59 OR L60 OR L61) AND L62 NOT L72



L59          221 SEA FILE=EMBASE ABB=ON  COLLECTIN/CT
L60          535 SEA FILE=EMBASE ABB=ON  SURFACTANT PROTEIN D/CT
L61          375 SEA FILE=EMBASE ABB=ON  SURFACTANT ASSOCIATED PROTEIN/CT
L63         5975 SEA FILE=EMBASE ABB=ON  CONTACT LENS/CT
L64         1416 SEA FILE=EMBASE ABB=ON  LACRIMAL FLUID/CT
L65          900 SEA FILE=EMBASE ABB=ON  ARTIFICIAL TEAR/CT
L76            0 SEA FILE=EMBASE ABB=ON  (L59 OR L60 OR L61) AND (L63 OR L64 OR
                 L65)



L59          221 SEA FILE=EMBASE ABB=ON  COLLECTIN/CT
L60          535 SEA FILE=EMBASE ABB=ON  SURFACTANT PROTEIN D/CT
L61          375 SEA FILE=EMBASE ABB=ON  SURFACTANT ASSOCIATED PROTEIN/CT
```

```
L67          932 SEA FILE=EMBASE ABB=ON  EYE PROTECTION/CT
L77            1 SEA FILE=EMBASE ABB=ON  (L59 OR L60 OR L61) AND L67



L59          221 SEA FILE=EMBASE ABB=ON  COLLECTIN/CT
L60          535 SEA FILE=EMBASE ABB=ON  SURFACTANT PROTEIN D/CT
L61          375 SEA FILE=EMBASE ABB=ON  SURFACTANT ASSOCIATED PROTEIN/CT
L62       302195 SEA FILE=EMBASE ABB=ON  EYE DISEASE+NT/CT
L68       132501 SEA FILE=EMBASE ABB=ON  EYE+NT/CT
L69        30005 SEA FILE=EMBASE ABB=ON  PSEUDOMONAS AERUGINOSA/CT
L70         4680 SEA FILE=EMBASE ABB=ON  INFECTION RESISTANCE/CT
L72          665 SEA FILE=EMBASE ABB=ON  (L59 OR L60 OR L61)(L)EC/CT
L80           50 SEA FILE=EMBASE ABB=ON  (L59 OR L60 OR L61)(L)(AD OR DO OR DV
                 OR CT OR DT OR PD OR PK)/CT
L81            3 SEA FILE=EMBASE ABB=ON  L80 AND (L62 OR (L68 OR L69 OR L70))
                 NOT L72


=> s l73,l77,l81 not l66

L145           4 (L73 OR L77 OR L81) NOT L66

=> fil wpix; d que l96; d que l101;d que l103

FILE 'WPIX' ENTERED AT 14:58:40 ON 04 OCT 2006
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FILE LAST UPDATED:          2 OCT 2006      <20061002/UP>
MOST RECENT DERWENT UPDATE:     200663        <200663/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,
    PLEASE VISIT:
 http://www.stn-international.de/training_center/patents/stn_guide.pdf <

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http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf <<<

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    INDEX ENHANCEMENTS PLEASE VISIT:
http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<
'BI ABEX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE


L85           51 SEA FILE=WPIX ABB=ON  COLLECTIN/BI,ABEX OR COLLECTINS/BI,ABEX
                 OR COLLAGEN LIKE LECTIN#/BI,ABEX
L87        93281 SEA FILE=WPIX ABB=ON  EYE?/BI,ABEX
L88        11427 SEA FILE=WPIX ABB=ON  OCULAR?/BI,ABEX
L89        23904 SEA FILE=WPIX ABB=ON  OPHTHALM?/BI,ABEX
L90         8316 SEA FILE=WPIX ABB=ON  CONTACT LENS?/BI,ABEX
L91         1028 SEA FILE=WPIX ABB=ON  KERATITIS/BI,ABEX
L93         4610 SEA FILE=WPIX ABB=ON  D09-C01A/MC
L94        17613 SEA FILE=WPIX ABB=ON  B14-N03/MC OR C14-N03/MC
L96            6 SEA FILE=WPIX ABB=ON  L85 AND (L87 OR L88 OR L89 OR L90 OR L91
                 OR L93 OR L94)
```

```
L87        93281 SEA FILE=WPIX ABB=ON  EYE?/BI,ABEX
L88        11427 SEA FILE=WPIX ABB=ON  OCULAR?/BI,ABEX
L89        23904 SEA FILE=WPIX ABB=ON  OPHTHALM?/BI,ABEX
L90         8316 SEA FILE=WPIX ABB=ON  CONTACT LENS?/BI,ABEX
L91         1028 SEA FILE=WPIX ABB=ON  KERATITIS/BI,ABEX
L93         4610 SEA FILE=WPIX ABB=ON  D09-C01A/MC
L94        17613 SEA FILE=WPIX ABB=ON  B14-N03/MC OR C14-N03/MC
L100          66 SEA FILE=WPIX ABB=ON  (SP/BI,ABEX OR SURFACTANT/BI,ABEX(1W)PROT
                 EIN/BI,ABEX)(W)D/BI,ABEX
L101           3 SEA FILE=WPIX ABB=ON  L100 AND (L87 OR L88 OR L89 OR L90 OR
                 L91 OR L93 OR L94)



L85           51 SEA FILE=WPIX ABB=ON  COLLECTIN/BI,ABEX OR COLLECTINS/BI,ABEX
                 OR COLLAGEN LIKE LECTIN#/BI,ABEX
L100          66 SEA FILE=WPIX ABB=ON  (SP/BI,ABEX OR SURFACTANT/BI,ABEX(1W)PROT
                 EIN/BI,ABEX)(W)D/BI,ABEX
L102       30746 SEA FILE=WPIX ABB=ON  TEAR#/BI,ABEX
L103           1 SEA FILE=WPIX ABB=ON  L102 AND (L100 OR L85)


=> s l96,l101,l103 not l92

L146           7 (L96 OR L101 OR L103) NOT L92

=> fil medl; d que l45; d que l48; d que l50; d que l52; d que l55

FILE 'MEDLINE' ENTERED AT 14:58:43 ON 04 OCT 2006

 FILE LAST UPDATED: 3 Oct 2006 (20061003/UP).  FILE COVERS 1950 TO DATE.

 On December 11, 2005, the 2006 MeSH terms were loaded.

 The MEDLINE reload for 2006 is now (26 Feb.) available.  For details
 on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).
 See also:

    http://www.nlm.nih.gov/mesh/
    http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

 OLDMEDLINE is covered back to 1950.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2006 vocabulary.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


L36         2293 SEA FILE=MEDLINE ABB=ON  COLLECTINS+NT/CT
L37       320667 SEA FILE=MEDLINE ABB=ON  EYE DISEASES+NT/CT
L43       221621 SEA FILE=MEDLINE ABB=ON  EYE+NT/CT
L44        22450 SEA FILE=MEDLINE ABB=ON  PSEUDOMONAS AERUGINOSA/CT
L45            1 SEA FILE=MEDLINE ABB=ON  (L43 OR L37) AND L44 AND L36
```

```
L36         2293 SEA FILE=MEDLINE ABB=ON  COLLECTINS+NT/CT
L37       320667 SEA FILE=MEDLINE ABB=ON  EYE DISEASES+NT/CT
L47          202 SEA FILE=MEDLINE ABB=ON  L36(L)(BL OR BI)/CT
L48            4 SEA FILE=MEDLINE ABB=ON  L47 AND L37


L36         2293 SEA FILE=MEDLINE ABB=ON  COLLECTINS+NT/CT
L37       320667 SEA FILE=MEDLINE ABB=ON  EYE DISEASES+NT/CT
L49         1219 SEA FILE=MEDLINE ABB=ON  ACANTHAMOEBA/CT
L50            1 SEA FILE=MEDLINE ABB=ON  L49 AND L36 AND L37


L36         2293 SEA FILE=MEDLINE ABB=ON  COLLECTINS+NT/CT
L41         4849 SEA FILE=MEDLINE ABB=ON  TEARS/CT
L52            1 SEA FILE=MEDLINE ABB=ON  L36 AND L41


L36         2293 SEA FILE=MEDLINE ABB=ON  COLLECTINS+NT/CT
L37       320667 SEA FILE=MEDLINE ABB=ON  EYE DISEASES+NT/CT
L43       221621 SEA FILE=MEDLINE ABB=ON  EYE+NT/CT
L44        22450 SEA FILE=MEDLINE ABB=ON  PSEUDOMONAS AERUGINOSA/CT
L54           89 SEA FILE=MEDLINE ABB=ON  L36(L)(AD OR PD OR PK OR TU)/CT
L55            4 SEA FILE=MEDLINE ABB=ON  L54 AND (L43 OR L44 OR L37)

=> s l45,l48,l50,l52,l55 not l42

L147           9 (L45 OR L48 OR L50 OR L52 OR L55) NOT L42

=> => dup rem l147,l144,l146,l145,l141
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PROCESSING COMPLETED FOR L147
PROCESSING COMPLETED FOR L144
PROCESSING COMPLETED FOR L146
PROCESSING COMPLETED FOR L145
PROCESSING COMPLETED FOR L141
L148 45 DUP REM L147 L144 L146 L145 L141 (17 DUPLICATES REMOVED)
ANSWERS '1-9' FROM FILE MEDLINE
ANSWERS '10-20' FROM FILE CAPLUS
ANSWERS '21-23' FROM FILE WPIX
ANSWERS '24-26' FROM FILE EMBASE
ANSWERS '27-28' FROM FILE JICST-EPLUS
ANSWER '29' FROM FILE CABA
ANSWERS '30-40' FROM FILE BIOSIS
ANSWERS '41-42' FROM FILE DISSABS
ANSWERS '43-45' FROM FILE SCISEARCH

=> d iall 1-9; d ibib ed abs hitind 10-20; d iall abeq tech 21-23; d iall 24-45; fil hom

L148 ANSWER 1 OF 45 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2005063671 MEDLINE Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PubMed ID: 15693089
TITLE: Serum mannose-binding lectin levels are decreased in behcet's disease and associated with disease severity.
AUTHOR: Inanc Nevsun; Mumcu Gonca; Birtas Elif; Elbir Yesim; Yavuz Sule; Ergun Tulin; Fresko Izzet; Direskeneli Haner
CORPORATE SOURCE: Division of Rheumatology and Hematology, Faculty of Medicine, University of Marmara, Istanbul, Turkey.. nevsun@superonline.com
SOURCE: The Journal of rheumatology, (2005 Feb) Vol. 32, No. 2, pp. 287-91.
Journal code: 7501984. ISSN: 0315-162X.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 5 Feb 2005
Last Updated on STN: 29 Apr 2005
Entered Medline: 28 Apr 2005
ABSTRACT:
OBJECTIVE: To investigate serum levels of mannose-binding lectin (MBL), a complement-like protein of collectin family, in patients with Behcet's disease (BD). METHODS: MBL levels were measured in sera of 130 patients with BD, 64 patients with recurrent oral ulcerations (ROU), and 105 healthy controls (HC) with ELISA. RESULTS: Patients with BD had significantly lower median serum MBL levels compared to HC (1857 vs 3136 ng/ml, p = 0.001). No significant difference was observed in median serum MBL levels between BD and ROU (2309 ng/ml, p = 0.252). Low MBL levels (</= 500 ng/ml) were present in a higher proportion of BD patients compared to HC (29% vs 16%, p = 0.021). A severe disease course (total clinical severity score >/= 4) was more frequently observed in BD patients with very low serum MBL levels (</= 100 ng/ml) (19% vs 6%, p = 0.046). When serum MBL levels were analyzed separately according to gender, the frequency of vascular disease was higher in men with very low serum

MBL levels (80% vs 42%, p = 0.042). CONCLUSIONS: MBL deficiency might contribute to the pathogenesis of BD and affect its clinical course.

CONTROLLED TERM: Check Tags: Female; Male
Adolescent
Adult
***Behcet Syndrome: BL, blood**
Behcet Syndrome: CO, complications
Behcet Syndrome: PA, pathology
Humans
***Mannose-Binding Lectin: BL, blood**
Middle Aged
Oral Ulcer: CO, complications
Oral Ulcer: PA, pathology
Research Support, Non-U.S. Gov't
*Severity of Illness Index

CHEMICAL NAME: 0 (Mannose-Binding Lectin)

L148 ANSWER 2 OF 45 MEDLINE on STN DUPLICATE 6

ACCESSION NUMBER: 2005314869 MEDLINE Full-text<<LOGINID::20061004>>

DOCUMENT NUMBER: PubMed ID: 15790974

TITLE: Defective surfactant secretion in a mouse model of Hermansky-Pudlak syndrome.

AUTHOR: Guttentag Susan H; Akhtar Amana; Tao Jian-Qin; Atochina Elena; Rusiniak Michael E; Swank Richard T; Bates Sandra R

CORPORATE SOURCE: Division of Neonatology, Department of Pediatrics, Children's Hospital of Philadelphia, 19104-4318, USA.. guttentag@email.chop.edu

CONTRACT NUMBER: CA-16056 (NCI)
EY-12104 (NEI)
HL-31698 (NHLBI)
HL-51480 (NHLBI)
HL56401 (NHLBI)
HL59959 (NHLBI)

SOURCE: American journal of respiratory cell and molecular biology, (2005 Jul) Vol. 33, No. 1, pp. 14-21. Electronic Publication: 2005-03-24. Journal code: 8917225. ISSN: 1044-1549.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200508

ENTRY DATE: Entered STN: 21 Jun 2005
Last Updated on STN: 3 Aug 2005
Entered Medline: 2 Aug 2005

ABSTRACT:
Hermansky-Pudlak syndrome (HPS) in humans represents a family of disorders of lysosome-related organelle biogenesis associated with severe, progressive pulmonary disease. Human case reports and a mouse model of HPS, the pale ear/pearl mouse (ep/pe), exhibit giant lamellar bodies (GLB) in type II alveolar epithelial cells. We examined surfactant proteins and phospholipid from ep/pe mice to elucidate the process of GLB formation. The 2.8-fold enrichment of tissue phospholipids in ep/pe mice resulted from accumulation from birth through adulthood. Tissue surfactant protein (SP)-B and -C were increased in adult ep/pe mice compared with wild-type mice (WT), whereas SP-A and -D were not different. Large aggregate surfactant (LA) from adult ep/pe mice had decreased phospholipid, SP-B, and SP-C, with no differences in SP-A and -D compared with WT. Although LA from ep/pe animals exhibited an increased total protein-to-total phospholipid ratio compared with WT, surface tension was not compromised. Phospholipid secretion from isolated type II cells showed

that basal and stimulated secretion from ep/pe cells were approximately 50% of WT cells. Together, our data indicate that GLB formation is not associated with abnormal trafficking or recycling of surfactant material. Instead, impaired secretion is an important component of GLB formation in ep/pe mice.

CONTROLLED TERM: Check Tags: Male
Animals
Blotting, Western
Bronchoalveolar Lavage
Capillaries: ME, metabolism
Densitometry
Disease Models, Animal
***Hermanski-Pudlak Syndrome: ME, metabolism**
***Hermanski-Pudlak Syndrome: PA, pathology**
Humans
Immunoblotting
Mice
Mice, Inbred C57BL
Mice, Transgenic
Microscopy, Fluorescence
Phospholipids: ME, metabolism
Pulmonary Surfactant-Associated Protein A: BI, biosynthesis
Pulmonary Surfactant-Associated Protein B: BI, biosynthesis
Pulmonary Surfactant-Associated Protein C: BI, biosynthesis
Pulmonary Surfactant-Associated Protein D: BI, biosynthesis
RNA, Messenger: ME, metabolism
Research Support, N.I.H., Extramural
Research Support, U.S. Gov't, P.H.S.
Reverse Transcriptase Polymerase Chain Reaction
*Surface-Active Agents: ME, metabolism
Time Factors

CHEMICAL NAME: 0 (Phospholipids); 0 (Pulmonary Surfactant-Associated Protein A); 0 (Pulmonary Surfactant-Associated Protein B); 0 (Pulmonary Surfactant-Associated Protein C); 0 (Pulmonary Surfactant-Associated Protein D); 0 (RNA, Messenger); 0 (Surface-Active Agents)

L148 ANSWER 3 OF 45 MEDLINE on STN
ACCESSION NUMBER: 2006117682 MEDLINE Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PubMed ID: 16505041
TITLE: In vitro pathogenicity of Acanthamoeba is associated with the expression of the mannose-binding protein.
AUTHOR: Garate Marco; Marchant Jeffrey; Cubillos Ibis; Cao Zhiyi; Khan Naveed A; Panjwani Noorjahan
CORPORATE SOURCE: Department of Ophthalmology, Center for Vision Research, Tufts University School of Medicine, Boston, Massachusetts 02111, USA.
CONTRACT NUMBER: EY09349 (NEI)
EYP30-13078
SOURCE: Investigative ophthalmology & visual science, (2006 Mar) Vol. 47, No. 3, pp. 1056-62.
Journal code: 7703701. ISSN: 0146-0404.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200604

ENTRY DATE: Entered STN: 1 Mar 2006
Last Updated on STN: 4 Apr 2006
Entered Medline: 3 Apr 2006

ABSTRACT:
PURPOSE: To determine whether the expression of Acanthamoeba mannose-binding protein (MBP) is associated with the pathogenicity of the parasite in vitro. METHODS: Both active trophozoites and dormant cysts of a pathogenic strain of A. castellanii were analyzed for their ability to bind to corneal epithelium, express MBP, and produce a cytopathic effect (CPE) on host cells. In addition, host cell binding, CPE-inducing ability, and MBP expression pattern of trophozoites of four different isolates of Acanthamoeba with various degrees of in vitro pathogenicity were analyzed. Binding assays were performed with radiolabeled parasites; CPE assays were performed with rabbit corneal epithelial cells as host cells; and the expression of MBP was detected by affinity chromatography of parasite extracts on mannose affinity columns and by immunohistochemical and Western blot analyses. RESULTS: Trophozoites of A. castellanii bound avidly to corneal epithelial cells in a mannose-inhibitable manner, whereas cysts exhibited little binding. The lack of binding of the cysts to host cells was associated with the downregulation of MBP, along with the concomitant loss of CPE. Analysis of trophozoites of five different species of Acanthamoeba exhibiting various degrees of pathogenic potential revealed that the ability of parasites to bind to host cells and produce CPE is directly correlated with the expression of the MBP. Acanthamoeba strains that bound avidly to host cells and produced potent CPE, robustly expressed MBP. In contrast, parasite strains that produced only weak CPE, expressed markedly reduced levels of MBP. CONCLUSIONS: The data demonstrating that the pathogenic potential of Acanthamoeba directly correlates with the expression level of the MBP in conjunction with our published studies showing that Acanthamoeba MBP is a major virulence protein suggest that the amoeba lectin has the potential to serve as a marker of pathogenicity.

CONTROLLED TERM:
***Acanthamoeba: ME, metabolism**
***Acanthamoeba: PY, pathogenicity**
***Acanthamoeba Keratitis: PS, parasitology**
Animals
Blotting, Western
Cell Adhesion: PH, physiology
Cells, Cultured
Chromatography, Affinity
*Epithelium, Corneal: PS, parasitology
***Mannose-Binding Lectin: ME, metabolism**
*Protozoan Proteins: ME, metabolism
Rabbits
Research Support, N.I.H., Extramural
Research Support, Non-U.S. Gov't

CHEMICAL NAME: 0 (Mannose-Binding Lectin); 0 (Protozoan Proteins)

L148 ANSWER 4 OF 45 MEDLINE on STN
ACCESSION NUMBER: 2005631293 MEDLINE Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PubMed ID: 16260422
TITLE: Thyroid hormone increases mannan-binding lectin levels.
AUTHOR: Riis Anne Lene Dalkjaer; Hansen Troels Krarup; Thiel Steffen; Gravholt Claus Hojbjerg; Gjedde Signe; Gormsen Lars Christian; Jorgensen Jens Otto Lunde; Weeke Jorgen; Moller Niels
CORPORATE SOURCE: Medical Department M, Aarhus University Hospital, Denmark.. anne.lene.riis@ki.au.dk
SOURCE: European journal of endocrinology / European Federation of Endocrine Societies, (2005 Nov) Vol. 153, No. 5, pp. 643-9. Journal code: 9423848. ISSN: 0804-4643.
PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
(CLINICAL TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200512
ENTRY DATE: Entered STN: 30 Nov 2005
Last Updated on STN: 22 Dec 2005
Entered Medline: 21 Dec 2005

ABSTRACT:
BACKGROUND: Recent studies have indicated the existence of causal links between the endocrine and immune systems and cardiovascular disease. Mannan-binding lectin (MBL), a protein of the innate immune system, may constitute a connection between these fields. METHODS: To test whether thyroid hormone regulates MBL levels, we studied eight patients with Graves' hyperthyroidism before and after methimazole therapy, eight healthy subjects before and after short-term experimental hyperthyroidism, and eight hypothyroid patients with chronic auto-immune thyroiditis before and after L-thyroxine substitution. RESULTS: In all hyperthyroid patients, MBL levels were increased--median (range), 1886 ng/ml (1478-7344) --before treatment and decreased to 954 ng/ml (312-3222) after treatment (P = 0.01, paired comparison: Wilcoxon's signed ranks test). The healthy subjects had MBL levels of 1081 ng/ml (312-1578). Administration of thyroid hormones to these persons induced mild hyperthyroidism and increased MBL levels significantly to 1714 ng/ml (356-2488) (P = 0.01). Two of the eight hypothyroid patients had undetectably low levels of MBL both before and after L-thyroxine substitution. The other six hypothyroid patients had decreased levels of MBL of 145 ng/ml (20-457) compared with 979 ng/ml (214-1533) after L-thyroxine substitution (P = 0.03, paired comparison: Wilcoxon's signed ranks test). CONCLUSION: Our data show that thyroid hormone increases levels of MBL. MBL is part of the inflammatory complement system, and this modulation of complement activation may play a role in the pathogenesis of a number of key components of thyroid diseases.

CONTROLLED TERM: Check Tags: Female; Male
Adult
Antithyroid Agents: TU, therapeutic use
***Graves Disease: BL, blood**
Graves Disease: DT, drug therapy
Humans
*Hyperthyroidism: BL, blood
Hyperthyroidism: CI, chemically induced
*Hypothyroidism: BL, blood
***Mannose-Binding Lectin: BL, blood**
Mannose-Binding Protein-Associated Serine Proteases: ME, metabolism
Methimazole: TU, therapeutic use
Middle Aged
Single-Blind Method
*Thyroid Hormones: PD, pharmacology
*Thyroiditis, Autoimmune: BL, blood
Thyroxine: PD, pharmacology
Triiodothyronine: PD, pharmacology

CAS REGISTRY NO.: 60-56-0 (Methimazole); 6893-02-3 (Triiodothyronine); 7488-70-2 (Thyroxine)

CHEMICAL NAME: 0 (Antithyroid Agents); 0 (Mannose-Binding Lectin); 0 (Thyroid Hormones); EC 3.4.21.- (MASP2 protein, human); EC 3.4.21.- (Mannose-Binding Protein-Associated Serine Proteases)

L148 ANSWER 5 OF 45 MEDLINE on STN
ACCESSION NUMBER: 2005525528 MEDLINE Full-text<<LOGINID::20061004>>

DOCUMENT NUMBER: PubMed ID: 16130114
TITLE: Agglutination of Pseudomonas aeruginosa by surfactant protein D.
AUTHOR: Griese Matthias; Starosta Vitaliy
CORPORATE SOURCE: Lung Research Group, Children's Hospital, Ludwig-Maximilians University, Munich, Germany.. matthias.griese@med.uni-muenchen.de
SOURCE: Pediatric pulmonology, (2005 Nov) Vol. 40, No. 5, pp. 378-84.
Journal code: 8510590. ISSN: 8755-6863.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200603
ENTRY DATE: Entered STN: 4 Oct 2005
Last Updated on STN: 17 Mar 2006
Entered Medline: 16 Mar 2006

ABSTRACT:
Surfactant protein D (SP-D) is part of the innate host defense system, and may bind and agglutinate invading microorganisms to enhance their removal. The ability of bronchoalveolar lavage (BAL) fluid to agglutinate bacteria and the relationship to its SP-D content are of interest and not yet known. A micromethod on slides was used to assess the agglutination of Pseudomonas aeruginosa by recombinant SP-D and native human BAL fluid. The SP-D-induced agglutination was blocked by calcium depletion, alkaline pH, or the presence of maltose. Twenty-three of 30 BAL fluids from outpatients carrying a chronic tracheostoma clearly agglutinated P. aeruginosa, which was completely inhibited by maltose. The extent of the agglutination correlated weakly to the concentration of SP-D in the BAL fluid, but not to that of SP-A. The functional property, i.e., the agglutination of P. aeruginosa by BAL fluid, was characterized and appeared related in part to the concentration of SP-D. Additional factors, such as the multimeric organization of SP-D, are likely to contribute to the agglutination of microorganisms by BAL or other body fluids. The assay presented will allow the systematic evaluation of small-volume samples for SP-D agglutinating ability from subjects with various lung diseases.

CONTROLLED TERM: Check Tags: Female; Male
Adolescent
Agglutination Tests: MT, methods
*Bronchoalveolar Lavage Fluid: IM, immunology
Bronchoalveolar Lavage Fluid: MI, microbiology
Calcium: ME, metabolism
Child
Child, Preschool
Edetic Acid: PD, pharmacology
Humans
Hydrogen-Ion Concentration
Infant
Maltose: PD, pharmacology
***Pseudomonas aeruginosa: IM, immunology**
*Pulmonary Surfactant-Associated Protein A: IM, immunology
Pulmonary Surfactant-Associated Protein A: PD, pharmacology
*Pulmonary Surfactant-Associated Protein D: IM, immunology
Reproducibility of Results
Research Support, Non-U.S. Gov't
Sweetening Agents: PD, pharmacology
CAS REGISTRY NO.: 60-00-4 (Edetic Acid); 69-79-4 (Maltose); 7440-70-2

(Calcium)
CHEMICAL NAME: 0 (Pulmonary Surfactant-Associated Protein A); 0 (Pulmonary Surfactant-Associated Protein D); 0 (Sweetening Agents)

L148 ANSWER 6 OF 45 MEDLINE on STN
ACCESSION NUMBER: 2004615733 MEDLINE Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PubMed ID: 15377498
TITLE: SP-A1 and SP-A2 variants differentially enhance association of Pseudomonas aeruginosa with rat alveolar macrophages.
AUTHOR: Mikerov Anatoly N; Umstead Todd M; Huang Weixiong; Liu Wenlei; Phelps David S; Floros Joanna
CORPORATE SOURCE: Dept. of Cellular and Molecular Physiology, The Pennsylvania State University College of Medicine, Hershey, PA 17033, USA.
CONTRACT NUMBER: HL-68947 (NHLBI)
SOURCE: American journal of physiology. Lung cellular and molecular physiology, (2005 Jan) Vol. 288, No. 1, pp. L150-8. Electronic Publication: 2004-09-17. Journal code: 100901229. ISSN: 1040-0605.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200502
ENTRY DATE: Entered STN: 20 Dec 2004
Last Updated on STN: 11 Feb 2005
Entered Medline: 10 Feb 2005

ABSTRACT:
Chronic airway inflammation caused by Pseudomonas aeruginosa is an important feature of cystic fibrosis (CF). Surfactant protein A (SP-A) enhances phagocytosis of P. aeruginosa. Two genes, SP-A1 and SP-A2, encode human SP-A. We hypothesized that genetically determined differences in the activity of SP-A1 and SP-A2 gene products exist. To test this, we studied association of a nonmucoid P. aeruginosa strain (ATCC 39018) with rat alveolar macrophages in the presence or absence of insect cell-expressed human SP-A variants. We used two trios, each consisting of SP-A1, SP-A2, and their coexpressed SP-A1/SP-A2 variants. We tested the 6A(2) and 6A(4) alleles (for SP-A1), the 1A(0) and 1A alleles (for SP-A2), and their respective coexpressed SP-A1/SP-A2 gene products. After incubation of alveolar macrophages with P. aeruginosa in the presence of the SP-A variants at 37 degrees C for 1 h, the cell association of bacteria was assessed by light microscopy analysis. We found 1) depending on SP-A concentration and variant, SP-A2 variants significantly increased the cell association more than the SP-A1 variants (the phagocytic index for SP-A1 was approximately 52-95% of the SP-A2 activity); 2) coexpressed variants at certain concentrations were more active than single gene products; and 3) the phagocytic index for SP-A variants was approximately 18-41% of the human SP-A from bronchoalveolar lavage. We conclude that human SP-A variants in vitro enhance association of P. aeruginosa with rat alveolar macrophages differentially and in a concentration-dependent manner, with SP-A2 variants having a higher activity compared with SP-A1 variants.
CONTROLLED TERM: Check Tags: Male
Alleles
Animals
Cells, Cultured
Comparative Study
Humans
Insects
Macrophages, Alveolar: DE, drug effects
*Macrophages, Alveolar: MI, microbiology
Pseudomonas aeruginosa: IP, isolation &

purification
*Pseudomonas aeruginosa: PH, physiology
*Pulmonary Surfactant-Associated Protein A: AA, analogs & derivatives
*Pulmonary Surfactant-Associated Protein A: GE, genetics
*Pulmonary Surfactant-Associated Protein A: PD, pharmacology
Rats
Rats, Sprague-Dawley
Research Support, U.S. Gov't, P.H.S.
*Variation (Genetics)

CHEMICAL NAME: 0 (Pulmonary Surfactant-Associated Protein A); 0 (SFTPA1 protein, human); 0 (SFTPA2 protein, human)

L148 ANSWER 7 OF 45 MEDLINE on STN
ACCESSION NUMBER: 2004295130 MEDLINE Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PubMed ID: 15195551
TITLE: Cytokine stimulation by Pseudomonas aeruginosa--strain variation and modulation by pulmonary surfactant.
AUTHOR: Bufler Philip; Schikor Daniela; Schmidt Bettina; Griese Matthias
CORPORATE SOURCE: Dr. von Haunersches Kinderspital, University of Munich, Lindwarmstr. D-80337 Munich, Germany.
SOURCE: Experimental lung research, (2004 Apr-May) Vol. 30, No. 3, pp. 163-79.
Journal code: 8004944. ISSN: 0190-2148.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 16 Jun 2004
Last Updated on STN: 7 Jul 2004
Entered Medline: 6 Jul 2004

ABSTRACT:
Pulmonary surfactant and its components are part of the first-line immune defense within the lung. Here the authors show that the surfactant protein (SP) SP-D, but not SP-A, agglutinates some clinical isolates of Pseudomonas aeruginosa and Stenotrophomonas maltophilia. No agglutination of Staphylococcus aureus or Burkholderia cepacia was observed. The SP-D-induced agglutination of P. aeruginosa was not dependent on a specific lipopolysaccharide (LPS) serotype. The authors also show that SP-D, but not SP-A, increased the tumor necrosis factor (TNF alpha) release from human monocytic cells in response to a subset of P. aeruginosa and P. aeruginosa LPS. A clinical preparation of surfactant (Alveofact) blocked the TNF alpha release from monocytic cells induced by P. aeruginosa or its LPS. SP-A reversed the inhibitory effect of Alveofact in 6/8 strains of P. aeruginosa and 2/9 preparations of P. aeruginosa LPS. SP-D did not significantly alter the TNF alpha production induced by vital P. aeruginosa in the presence of Alveofact but markedly increased the TNF alpha release induced by a preparation of rough and smooth P. aeruginosa LPS. In summary, this study shows that the immunomodulatory properties of SP-A and SP-D specifically depend on the colonizing strain of P. aeruginosa. In addition, the authors show that the function of SP-A and SP-D is modulated in the presence of surfactant lipids.

CONTROLLED TERM: Bacterial Adhesion: DE, drug effects
Cell Line
Cystic Fibrosis: IM, immunology
Down-Regulation: DE, drug effects
Humans
Lipids: PD, pharmacology

Lipopolysaccharides: PD, pharmacology
Monocytes: CY, cytology
Monocytes: ME, metabolism
*Monocytes: MI, microbiology
Phospholipids: PD, pharmacology
Pseudomonas Infections: IM, immunology
*Pseudomonas Infections: ME, metabolism
Pseudomonas aeruginosa: CL, classification
***Pseudomonas aeruginosa: IM, immunology**
***Pulmonary Surfactant-Associated Protein A: PD, pharmacology**
Pulmonary Surfactant-Associated Protein D: PD, pharmacology
Research Support, Non-U.S. Gov't
Species Specificity
*Tumor Necrosis Factor-alpha: ME, metabolism

CHEMICAL NAME: 0 (Lipids); 0 (Lipopolysaccharides); 0 (Phospholipids); 0 (Pulmonary Surfactant-Associated Protein A); 0 (Pulmonary Surfactant-Associated Protein D); 0 (SF-RI 1, bovine surfactant preparation); 0 (Tumor Necrosis Factor-alpha)

L148 ANSWER 8 OF 45 MEDLINE on STN
ACCESSION NUMBER: 2003382608 MEDLINE Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PubMed ID: 12918709
TITLE: Low serum mannose-binding lectin levels in Behcet's disease.
AUTHOR: Inanc Nevsun; Birtas Elif; Yavuz Sule; Ergun Tulin; Direskeneli Haner
CORPORATE SOURCE: Department of Internal Medicine, Faculty of Medicine, Marmara University, Istanbul, Turkey.
SOURCE: Advances in experimental medicine and biology, (2003) Vol. 528, pp. 287-9.
Journal code: 0121103. ISSN: 0065-2598.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 16 Aug 2003
Last Updated on STN: 18 Dec 2003
Entered Medline: 2 Dec 2003
CONTROLLED TERM: Check Tags: Female; Male
Adult
Arthritis, Rheumatoid: BL, blood
Arthritis, Rheumatoid: IM, immunology
***Behcet Syndrome: BL, blood**
***Behcet Syndrome: IM, immunology**
Humans
***Mannose-Binding Lectin: BL, blood**
Middle Aged
Oral Ulcer: BL, blood
Oral Ulcer: IM, immunology
Recurrence
Reference Values
CHEMICAL NAME: 0 (Mannose-Binding Lectin)

L148 ANSWER 9 OF 45 MEDLINE on STN
ACCESSION NUMBER: 2003033473 MEDLINE Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PubMed ID: 12540493
TITLE: Surfactant protein A and D differently regulate the immune

response to nonmucoid Pseudomonas aeruginosa and its lipopolysaccharide.

AUTHOR: Bufler Philip; Schmidt Bettina; Schikor Daniela; Bauernfeind Adolf; Crouch Erika C; Griese Matthias
CORPORATE SOURCE: Dr. von Haunersches Kinderspital, University of Munich, Munich, Germany.

SOURCE: American journal of respiratory cell and molecular biology, (2003 Feb) Vol. 28, No. 2, pp. 249-56.
Journal code: 8917225. ISSN: 1044-1549.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 24 Jan 2003
Last Updated on STN: 2 Mar 2003
Entered Medline: 28 Feb 2003

ABSTRACT:
We investigated the role of the surfactant proteins (SPs) A and D in the pulmonary immune defense of nonmucoid strains of Pseudomonas aeruginosa, the most etiologic agents of nosocomial Pseudomonas pneumonia. We first examined the interactions of recombinant human SP-D dodecamers and purified natural or recombinant human SP-A with two smooth, and two rough, clinical isolates of nonmucoid P. aeruginosa. SP-D bound to all four isolates, but agglutinated only one rough and one smooth strain. SP-D functioned as an opsonin to enhance the uptake of all four strains by the human monocytic cell line Mono Mac 6 (MM6). SP-D also enhanced tumor necrosis factor-alpha secretion by MM6 cells in response to purified lipopolysaccharide (LPS) isolated from the rough, but not the smooth, strains. Although SP-A bound to all four strains, it did not cause bacterial aggregation or enhance uptake. It showed small but statistically significant inhibitory effects on the cytokine response of MM6 cells to one strain of smooth organisms, but did not significantly alter the response to purified LPS. This study in combination with previously published data strongly suggests that SP-D may play important roles in the local innate pulmonary defense against nonmucoid P. aeruginosa of diverse LPS phenotypes, and preferentially augments the cellular response to rough P. aeruginosa endotoxin.

CONTROLLED TERM:
Animals
Cell Line
Humans
*Lipopolysaccharides: IM, immunology
Mice
Monocytes: DE, drug effects
Monocytes: IM, immunology
Phagocytosis
Pneumonia, Bacterial: ET, etiology
Pneumonia, Bacterial: IM, immunology
Pseudomonas Infections: ET, etiology
Pseudomonas Infections: IM, immunology
***Pseudomonas aeruginosa: IM, immunology**
Pseudomonas aeruginosa: IP, isolation & purification
Pseudomonas aeruginosa: PY, pathogenicity
*Pulmonary Surfactant-Associated Protein A: IM, immunology
Pulmonary Surfactant-Associated Protein A: PD, pharmacology
*Pulmonary Surfactant-Associated Protein D: IM, immunology
Pulmonary Surfactant-Associated Protein D: PD, pharmacology
Recombinant Proteins: IM, immunology

Recombinant Proteins: PD, pharmacology
Research Support, Non-U.S. Gov't
Tumor Necrosis Factor-alpha: BI, biosynthesis

CHEMICAL NAME: 0 (Lipopolysaccharides); 0 (Pulmonary Surfactant-Associated Protein A); 0 (Pulmonary Surfactant-Associated Protein D); 0 (Recombinant Proteins); 0 (Tumor Necrosis Factor-alpha)

L148 ANSWER 10 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2005:120697 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 142:183507
TITLE: Remedy or diagnostic for inflammatory disease containing target-directingliposome having sugar chain
INVENTOR(S): Yamazaki, Noboru; Tsurushima, Hideo; Ooguro, Nobuyuki
PATENT ASSIGNEE(S): National Institute of Advanced Industrial Science and Technology, Japan
SOURCE: PCT Int. Appl., 105 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005011633	A1	20050210	WO 2004-JP11329	20040730
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1655022	A1	20060510	EP 2004-771330	20040730
R: CH, DE, FR, GB, LI				

PRIORITY APPLN. INFO.:

JP 2003-285422	A	20030801
JP 2003-369494	A	20031029
WO 2004-JP11329	W	20040730

ED Entered STN: 11 Feb 2005

AB It is intended to provide target-directing drug delivery system (DDS) nanoparticles which accumulate in a target tissue such as an inflammation site in an inflammatory disease and thus are usable as a DDS for therapeutic or diagnostic use aiming at topically supplying a drug or a gene to the affected part. A medicinal composition for treating or diagnosing an inflammatory disease containing sugar chain-modified liposomes in which a sugar chain is bonded to liposome membrane. Prednisolone phosphate-containing original liposomes were prepared from dipalmitoylphosphatidylcholine, cholesterol, dicetylphosphate, ganglioside, dipalmitoylphosphatidylethanol amine (at 35:40:5:15:5), and prednisolone phosphate. The obtained original liposomes were hydrophilized, bound with human serum albumin and an sialyl Lewis X tetrasaccharide glycosylamine compound with crosslinking substances, and then hydrophilized again to obtain inflammatory site-targeting liposomes having sugar chain through albumin.

IC ICM A61K009-127

ICS A61K047-24; A61K047-26; A61K047-28; A61K047-36; A61K047-42; A61K049-00; A61P029-00
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 9
IT Agglutinins and Lectins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**collectin**; target-directed and enteric absorption-controlled liposome having sugar chain)
IT **Eye, disease**
Inflammation
(**ophthalmitis**; target-directed and enteric absorption-controlled liposome having sugar chain)
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L148 ANSWER 11 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2005:120696 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 142:183506
TITLE: Target-directed and enteric absorption-controlled liposome having sugar chain and cancer remedy and diagnostic containing the same
INVENTOR(S): Yamazaki, Noboru; Tsurushima, Hideo; Kojima, Shuuji
PATENT ASSIGNEE(S): National Institute of Advanced Industrial Science and Technology, Japan
SOURCE: PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005011632	A1	20050210	WO 2004-JP11291	20040730
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1655038	A1	20060510	EP 2004-748262	20040730
R: CH, DE, FR, GB, LI				

PRIORITY APPLN. INFO.:	JP 2003-285432	A	20030801
	JP 2004-93872	A	20040326
	WO 2004-JP11291	W	20040730

OTHER SOURCE(S): MARPAT 142:183506
ED Entered STN: 11 Feb 2005
AB It is intended to provide a liposome having a sugar chain that has an activity of specifically binding to various lectins (sugar chain-recognizing proteins) existing on the cell surface in various tissues whereby a cell or a tissue in vivo can be distinguished in practice and thus a drug or a gene can be efficiently delivered thereto. Doxorubicin-containing original liposomes were prepared from dipalmitoylphosphatidylcholine, cholesterol, dicetylphosphate, ganglioside, dipalmitoylphosphatidylethanolamine (at 35:40:5:15:5), and doxorubicin. The obtained original liposomes were hydrophilized, bound with

human serum albumin and an α-1,6-mannobiose disaccharide glycosylamine compound with crosslinking substances, and then hydrophilized again to obtain tumor targeting liposomes having sugar chain through albumin.

IC ICM A61K009-127
ICS A61K047-24; A61K047-26; A61K047-28; A61K047-36; A61K047-42; A61K007-00; A61P035-00; A23L001-302; A23L001-00

CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1, 9, 17, 18, 62

IT **Eye, disease**
Urinary system
(agents for; target-directed and enteric absorption-controlled liposome having sugar chain)

IT Agglutinins and Lectins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**collectin**; target-directed and enteric absorption-controlled liposome having sugar chain)

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L148 ANSWER 12 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 9
ACCESSION NUMBER: 2003:335134 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 138:348719
TITLE: Nucleic acid-binding fragments of surfactant protein D for use in the treatment of inflammatory lung diseases
INVENTOR(S): Clark, Howard; Nadesalingam, Palaniyar; Reid, Kenneth Bannerman Milne; Strong, Peter
PATENT ASSIGNEE(S): Medical Research Council, UK
SOURCE: PCT Int. Appl., 167 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003035679	A2	20030501	WO 2002-GB4824	20021025
WO 2003035679	A3	20030731		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1440083	A2	20040728	EP 2002-772550	20021025
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005522988	T2	20050804	JP 2003-538192	20021025
US 2004259201	A1	20041223	US 2004-830959	20040423

PRIORITY APPLN. INFO.:
GB 2001-25638 A 20011025
GB 2002-9619 A 20020426
WO 2002-GB4824 W 20021025

ED Entered STN: 02 May 2003

AB A fragment of pulmonary surfactant protein D that binds nucleic acids and that is of therapeutic use in the treatment of pulmonary disease including asthma is described. A method of treating an individual suffering from a disease or

preventing the occurrence of a disease in an individual is also described, in which the method comprises administering to the individual a therapeutically or prophylactically effective amount of an rSPD(n/CRD) polypeptide, fragment, homolog, variant or derivative thereof. A 175-amino acid C-terminal fragment of the protein including the carbohydrate-binding domain was in Escherichia coli and purified by solubilization and renaturation of inclusion bodies and affinity chromatog. against maltose agarose. Itranasally delivered protein was able to limit the hypersensitive response to Aspergillus fumigatus antigens in surfactant protein D-deficient mice measured by serum IgE and IgG1 levels and peripheral eosinophilia.

IC ICM C07K014-00

CC 1-9 (Pharmacology)
Section cross-reference(s): 3, 6, 15, 17

IT Antibodies and Immunoglobulins
RL: ADV (Adverse effect, including toxicity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(IgE, therapeutic control of levels of; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of inflammatory lung diseases)

IT Antibodies and Immunoglobulins
RL: ADV (Adverse effect, including toxicity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(IgG1, therapeutic control of levels of; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of inflammatory lung diseases)

IT **Surfactant proteins** (pulmonary)
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(SP-B, in delivery of pulmonary **surfactant protein** D to lungs; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of inflammatory lung diseases)

IT **Surfactant proteins** (pulmonary)
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(SP-C, in delivery of pulmonary **surfactant protein** D to lungs; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of inflammatory lung diseases)

IT **Surfactant proteins** (pulmonary)
RL: PRP (Properties); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**SP-D**; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of inflammatory lung diseases)

IT **Eye, disease**
(allergic, treatment of; nucleic acid-binding fragments of surfactant protein D for use in treatment of inflammatory lung **diseases**)

IT Alcohols, biological studies
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(cetyl, in delivery of pulmonary **surfactant protein** D to lungs; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of inflammatory lung diseases)

IT Fatty acids, biological studies
Phospholipids, biological studies
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(in delivery of pulmonary **surfactant protein** D to lungs; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of inflammatory lung diseases)

IT 519065-05-5
RL: BSU (Biological study, unclassified); PRP (Properties); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(amino acid sequence; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of

inflammatory lung diseases)
IT 57-10-3, Palmitic acid, biological studies 63-89-8, Colfosceril palmitate 555-44-2, Tripalmitin 7647-14-5, Sodium chloride, biological studies 25301-02-4, Tyloxapol 129069-19-8, Poractant alfa
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(in delivery of pulmonary **surfactant protein** D to lungs; nucleic acid-binding fragments of **surfactant protein** D for use in treatment of inflammatory lung diseases)

L148 ANSWER 13 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 10
ACCESSION NUMBER: 2003:1007590 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 140:47549
TITLE: Amniotic membrane-mediated delivery of bioactive molecules
INVENTOR(S): Zhang, Fen
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 28 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003235580	A1	20031225	US 2003-603385	20030624
WO 2004000164	A2	20031231	WO 2003-US20021	20030624
WO 2004000164	A3	20041118		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003243781	A1	20040106	AU 2003-243781	20030624
PRIORITY APPLN. INFO.:			US 2002-391550P P	20020624
			WO 2003-US20021 W	20030624

ED Entered STN: 28 Dec 2003
AB The present invention provides reconstituted and recombinant tissue membranes and methods for pharmaceutical delivery of bioactive mols. In particular, reconstituted and recombinant amniotic membranes are provided for sustained delivery of therapeutic mols., proteins or metabolites, to a site of a host in need thereof. The reconstituted and recombinant amniotic membrane contains one or more recombinant expression vectors that are exogenous to the membrane and capable of expressing bioactive mols. The reconstituted and recombinant tissue membranes and methods can be used for in situ delivery of therapeutic proteins to a host in the treatment of disorders such as chronic wounds and dermatol. or ocular surface diseases. For example, growth factor PDGF-β, delivered by prototypic reconstituted amniotic membrane, promoted healing of ischemic wounds on rabbit ears as a model of the ischemic conditions of human chronic wounds.
IC ICM A61K039-395
INCL 424130100
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1, 3, 62
IT Agglutinins and Lectins

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**collectin**; amniotic membrane-mediated delivery of bioactive mols. to skin)

IT **Eye, disease**
(treatment of; amniotic membrane-mediated delivery of bioactive mols. to skin and **eye**)

L148 ANSWER 14 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:183045 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 140:234386
TITLE: Chimeric proteins comprising lectin carbohydrate-binding domain and cell surface protein ligand for modulating immune response to antigen
INVENTOR(S): Segal, Andrew H.; Young, Elihu
PATENT ASSIGNEE(S): Genitrix, LLC, USA
SOURCE: PCT Int. Appl., 265 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004018698	A2	20040304	WO 2003-US26072	20030820
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004039156	A1	20040226	US 2002-224661	20020820
CA 2496384	AA	20040304	CA 2003-2496384	20030820
AU 2003265523	A1	20040311	AU 2003-265523	20030820
US 2004091503	A1	20040513	US 2003-645000	20030820
EP 1573047	A2	20050914	EP 2003-793170	20030820
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006517512	T2	20060727	JP 2004-531131	20030820
US 2004122217	A1	20040624	US 2003-666871	20030919
US 2004126793	A1	20040701	US 2003-666885	20030919
US 2004126357	A1	20040701	US 2003-666886	20030919
US 2004142889	A1	20040722	US 2003-666898	20030919
US 2004151728	A1	20040805	US 2003-666834	20030919
US 2004170960	A1	20040902	US 2003-667193	20030919
US 2004180389	A1	20040916	US 2003-667166	20030919
US 2004241137	A1	20041202	US 2003-666833	20030919
US 2005064391	A1	20050324	US 2003-668073	20030919
PRIORITY APPLN. INFO.:			US 2002-224661 A	20020820
			US 2002-404823P P	20020820
			US 2003-487407P P	20030715
			US 2003-645000 A3	20030820
			WO 2003-US26072 W	20030820

ED Entered STN: 05 Mar 2004
AB The present invention provides a fusion polypeptide which can bind to a cell surface binding moiety (e.g., a carbohydrate) and server as a ligand for a

cell surface polypeptide, as well as a vector comprising a nucleic acid encoding for such a fusion polypeptide, and a host cell comprising such nucleic acid. The lectin is collectin, galectin, C-type lectin or glycoprotein; and the cell surface protein is cytokine receptor, CD40, adhesion mol., defensin receptor, heat shock protein receptor, T cell costimulatory mol., counterreceptor of T cell costimulatory mol., or opsonin receptor. The present invention also provides a composition comprising an antigen bearing target and such a fusion polypeptide, as well as a composition comprising a virus or a cell and such a fusion polypeptide. The antigen is tumor antigen, viral antigen, bacterial antigen, fungal antigen, parasitic antigen, prion antigen, or autoimmune disease antigen. The present invention further relates to a method of modulating an immune response in an animal using such compns. or vaccines.

IC ICM C12Q

CC 15-2 (Immunochemistry)
Section cross-reference(s): 3

IT Agglutinins and Lectins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**collectin**; chimeric proteins comprising lectin carbohydrate-binding domain and cell surface protein ligand for enhancing immune response to vaccines against infection, cancer, prion disease and autoimmune disease)

IT **Eye**, neoplasm
(retinoblastoma; chimeric proteins comprising lectin carbohydrate-binding domain and cell surface protein ligand for enhancing immune response to vaccines against infection, cancer, prion **disease** and autoimmune **disease**)

L148 ANSWER 15 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:266918 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 140:282485
TITLE: Methods for diagnosing interstitial lung diseases using biomarkers identified by microarray gene expression profiling
INVENTOR(S): Bevec, Dorian
PATENT ASSIGNEE(S): Mondobiotech SA, Switz.
SOURCE: Eur. Pat. Appl., 43 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1403638	A1	20040331	EP 2002-21413	20020925
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRIORITY APPLN. INFO.:			EP 2002-21413	20020925

ED Entered STN: 01 Apr 2004

AB The present invention relates to mol. methods diagnosing interstitial lung diseases (ILDs) using microarrays of candidate polynucleotides. The present invention also relates to methods useful in mol. evaluation of the efficacy of a drug applied to a person in need suffering from an ILD by gene expression profiling images. An aspect of the invention relates to the use of polynucleotide arrays, which allows to quant. study mRNA expression levels of selected candidate genes in human biopsies. A method for detecting gene expression of infective agents from patients with ILD is also disclosed.

IC ICM G01N033-48

CC 3-4 (Biochemical Genetics)
Section cross-reference(s): 1, 14
IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); DGN (Diagnostic use); **THU (Therapeutic use)**;
ANST (Analytical study); BIOL (Biological study); USES (Uses)
(SP-A, A2, NM_006926; methods for diagnosing interstitial lung diseases using biomarkers identified by microarray gene expression profiling)
IT Proteins
RL: ANT (Analyte); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**eyes** absent (Drosophila) homolog 2, U71207; methods for diagnosing interstitial lung **diseases** using biomarkers identified by microarray gene expression profiling)
IT **Proteins**
RL: ANT (Analyte); DGN (Diagnostic use); **THU (Therapeutic use)**;
ANST (Analytical study); BIOL (Biological study); USES (Uses)
(pulmonary **surfactant protein** (SP5), J03553; methods for diagnosing interstitial lung diseases using biomarkers identified by microarray gene expression profiling)
IT **Proteins**
RL: ANT (Analyte); DGN (Diagnostic use); **THU (Therapeutic use)**;
ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**surfactant**, pulmonary-associated **protein** C, BC005913; methods for diagnosing interstitial lung diseases using biomarkers identified by microarray gene expression profiling)
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L148 ANSWER 16 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:780650 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 135:335149
TITLE: Particulate compositions based on crosslinked polymers
INVENTOR(S): Dickinson, Paul Alfred; Kellaway, Ian Walter; Howells, Stephen Wyn
PATENT ASSIGNEE(S): University College Cardiff Consultants Limited, UK
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001078689	A2	20011025	WO 2001-GB1752	20010418
WO 2001078689	A3	20020328		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2405659	AA	20011025	CA 2001-2405659	20010418
EP 1274403	A2	20030115	EP 2001-921626	20010418
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003161886	A1	20030828	US 2003-258190	20030117

US 7018657 B2 20060328
US 2006093557 A1 20060504 US 2005-305784 20051216
PRIORITY APPLN. INFO.: GB 2000-9773 A 20000419
WO 2001-GB1752 W 20010418
US 2003-258190 A1 20030117

ED Entered STN: 26 Oct 2001

AB Nanoparticles are prepared from a colloidal system comprising a continuous phase and micelles, the micelles comprising surfactant material. A microemulsion is formed by admixing the colloidal system with a solution of an active material, such as a medicament, dissolved in a solvent wherein the solution forms a disperse phase with the micelles of surfactant material. At least the dispersed phase is quenched to a solid state and the continuous phase and solvent are removed to produce the nanoparticles. The nanoparticles can be incorporated in an aerosol composition suitable for deep lung delivery by means of a metered dose inhaler. For example, nanoparticles were formed using iso-octane, the lecithin/propanol-2-ol (1:3 by weight) surfactant system including as the active material pEGFP-N1 reporter plasmid DNA (4700 base pairs). The particles also contained protamine sulfate (1:1 by weight with respect to pDNA) and sucrose at a concentration of 0.5M in the aqueous phase.

IC ICM A61K009-51
ICS A61K009-12

CC 63-6 (Pharmaceuticals)

IT Drug delivery systems
(**ophthalmic**; preparation of crosslinked polymer nanoparticles from colloidal system comprising continuous phase and surfactant micelles)

IT Alkyl chlorides
Bile salts
Carbohydrates, biological studies
Corticosteroids, biological studies
DNA
Disaccharides
Monosaccharides
Nucleic acids
Peptides, biological studies
Phospholipids, biological studies
Proteins, general, biological studies
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(preparation of crosslinked polymer nanoparticles from colloidal system comprising continuous phase and **surfactant** micelles)

L148 ANSWER 17 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:661215 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 135:231732
TITLE: Nanocapsule encapsulation system based on surfactant micelles and polymers
INVENTOR(S): Unger, Gretchen M.
PATENT ASSIGNEE(S): Genesegues, Inc., USA
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001064164	A2	20010907	WO 2001-US6455	20010228
WO 2001064164	A3	20011220		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN,

IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2400172	AA	20010907	CA 2001-2400172		20010228
AU 2001047244	A5	20010912	AU 2001-47244		20010228
EP 1267946	A2	20030102	EP 2001-920161		20010228
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
JP 2003524654	T2	20030819	JP 2001-563063		20010228
US 2003170893	A1	20030911	US 2001-796575		20010228
US 6632671	B2	20031014			
US 2004137071	A1	20040715	US 2003-652814		20030829
PRIORITY APPLN. INFO.:			US 2000-185282P	P	20000228
			US 2001-796575	A1	20010228
			WO 2001-US6455	W	20010228

ED Entered STN: 10 Sep 2001

AB The present invention generally relates to nanocapsules and methods of preparing these nanocapsules. The present invention includes a method of forming a surfactant micelle and dispersing the surfactant micelle into an aqueous composition having a hydrophilic polymer to form a stabilized dispersion of surfactant micelles. The method further includes mech. forming droplets of the stabilized dispersion of surfactant micelles, precipitating the hydrophilic polymer to form precipitated nanocapsules, incubating the nanocapsules to reduce a diameter of the nanocapsules, and filtering or centrifuging the nanocapsules. Nanocapsules are suitable for drug targeting, especially targeting of DNA in gene therapy. For example, the importance of appropriate dispersion conditions was investigated in the following series of formulations produced by (i) predispersing 25 μg of DNA on ice using a bath sonicator, (ii) condensing DNA in a small amount of water by vortexing then incubating on ice for 20 min, (iii) adding surfactant then oil followed by 30 s of probe sonication at 10 W, (iv) diffusion dilution to 3 mL (mL) by first adding saline then hyaluronan polymer (1%) as a protective colloid, (v) mech. shearing emulsion into droplets by pumping through a 250 μm orifice into 22 mL of PBS, 10 mM Ca2+, 200 mM Li+, (vi) incubating overnight end over end, and (vii) centrifuging to recover nanoparticles for resuspension and filter sterilization. The condenser-to-DNA weight ratio was determined by dye exclusion at 90% charge neutralization. 2,4,7,9-Tetramethyl-5-decyn-4,7-diol was used in this experiment to represent water-immiscible surfactant, while Tergitol NP 40 and Tween 80 were used to represent water-soluble and even more water-soluble emulsifiers/dispersing aids. Dispersion conditions were systematically varied to discourage micelle formation in aqueous media by (i) choosing water-soluble surfactants, (ii) removing the dispersed phase, and (iii) decreasing surfactant loading below that required for micelle formation. One formulation featured use of a water-miscible oil (silicone oil). Formulations were characterized phys. and tested for functionality in in vitro gene transfer.

IC ICM A61K

CC 63-7 (Pharmaceuticals)
Section cross-reference(s): 1, 3, 62

IT Artery
Eye
Liver
Urethra
(delivery to; nanocapsule encapsulation system based on surfactant micelles and polymers for gene therapy)

IT Castor oil

Mucins
Nucleic acids
Oligonucleotides
Phosphorothioate oligodeoxyribonucleotides
Polynucleotides
Polysiloxanes, biological studies
Promoter (genetic element)
Proteins, general, biological studies
Tenascins
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(nanocapsule encapsulation system based on **surfactant** micelles and polymers for gene therapy)

IT **Proteins**, specific or class
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(nuclear matrix-associated, conjugates with nuclear signal localization peptides; nanocapsule encapsulation system based on **surfactant** micelles and polymers for gene therapy)

L148 ANSWER 18 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:795994 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 132:31744
TITLE: Gene probes used for genetic profiling in healthcare screening and planning
INVENTOR(S): Roberts, Gareth Wyn
PATENT ASSIGNEE(S): Genostic Pharma Ltd., UK
SOURCE: PCT Int. Appl., 745 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9964627	A2	19991216	WO 1999-GB1780	19990604

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:		
GB 1998-12099	A	19980606
GB 1998-13291	A	19980620
GB 1998-13611	A	19980624
GB 1998-13835	A	19980627
GB 1998-14110	A	19980701
GB 1998-14580	A	19980707
GB 1998-15438	A	19980716
GB 1998-15574	A	19980718
GB 1998-15576	A	19980718
GB 1998-16085	A	19980724
GB 1998-16086	A	19980724
GB 1998-16921	A	19980805
GB 1998-17097	A	19980807
GB 1998-17200	A	19980808
GB 1998-17632	A	19980814
GB 1998-17943	A	19980819

ED Entered STN: 17 Dec 1999

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide critical clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic" profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

IC ICM C12Q001-68
ICS C07K016-18

CC 3-1 (Biochemical Genetics)
Section cross-reference(s): 9, 13, 14

IT **Proteins, specific or class**
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(Bagpipe homeobox, core group of **disease**-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT Antigens
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**CD139**, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT Antigens
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(CD160, core group of **disease**-related genes; gene probes used for **genetic** profiling in healthcare screening and planning)

IT **Antigens**
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**CD78**, core group of **disease**-related genes; gene probes used for genetic profiling in **healthcare** screening and planning)

IT **CD antigens**
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(CD83, core group of **disease**-related **genes**; gene probes used for genetic profiling in healthcare screening and planning)

IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); **THU (Therapeutic use)**; ANST (Analytical

study); BIOL (Biological study); USES (Uses)
(SP-A, A1 and A2, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); **THU (Therapeutic use)**; ANST (Analytical study); BIOL (Biological study); USES (Uses)
(SP-B, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); **THU (Therapeutic use)**; ANST (Analytical study); BIOL (Biological study); USES (Uses)
(SP-C, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); **THU (Therapeutic use)**; ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**SP-D**, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Eye, disease**
(achromotopsia gene ACHM2, core group of **disease**-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Eye, disease**
(choroideremia, gene CHM, core group of **disease**-related genes; gene probes used for genetic profiling in healthcare screening and planning)

L148 ANSWER 19 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:795993 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 132:31743
TITLE: Gene probes used for genetic profiling in healthcare screening and planning
INVENTOR(S): Roberts, Gareth Wyn
PATENT ASSIGNEE(S): Genostic Pharma Limited, UK
SOURCE: PCT Int. Appl., 149 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9964626	A2	19991216	WO 1999-GB1779	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2330929	AA	19991216	CA 1999-2330929	19990604
AU 9941586	A1	19991230	AU 1999-41586	19990604
AU 766544	B2	20031016		
AU 9941587	A1	19991230	AU 1999-41587	19990604
GB 2339200	A1	20000119	GB 1999-12914	19990604
GB 2339200	B2	20010912		
EP 1084273	A1	20010321	EP 1999-925207	19990604

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

JP 2003528564	T2	20030930	JP 2000-553616		19990604
US 2003198970	A1	20031023	US 2002-206568		20020729
PRIORITY APPLN. INFO.:			GB 1998-12098	A	19980606
			GB 1998-28289	A	19981223
			GB 1998-16086	A	19980724
			GB 1998-16921	A	19980805
			GB 1998-17097	A	19980807
			GB 1998-17200	A	19980808
			GB 1998-17632	A	19980814
			GB 1998-17943	A	19980819
			US 1999-325123	B1	19990603
			WO 1999-GB1779	W	19990604

ED Entered STN: 17 Dec 1999

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide critical clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

IC ICM C12Q001-68
ICS C07K016-18

CC 3-1 (Biochemical Genetics)
Section cross-reference(s): 9, 13, 14

IT **Proteins, specific or class**
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(Apaf-1, core group of **disease**-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT Antigens
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**CD12**, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT Antigens
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(CD138, core group of **disease**-related genes; gene probes used for **genetic** profiling in healthcare screening and planning)

IT **Glycoproteins, specific or class**
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(CD40-L (antigen CD40 ligand), core group of **disease**-related genes; **gene** probes used for genetic profiling in healthcare screening and planning)

IT **Antigens**
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**CD42**, core group of **disease**-related genes;

gene probes used for genetic profiling in healthcare screening and planning)

IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); **THU (Therapeutic use)**; ANST (Analytical study); BIOL (Biological study); USES (Uses)
(SP-A, A1 and A2, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); **THU (Therapeutic use)**; ANST (Analytical study); BIOL (Biological study); USES (Uses)
(SP-B, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); **THU (Therapeutic use)**; ANST (Analytical study); BIOL (Biological study); USES (Uses)
(SP-C, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Surfactant proteins** (pulmonary)
RL: ANT (Analyte); **THU (Therapeutic use)**; ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**SP-D**, core group of disease-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Eye, disease**
(achromotopsia gene ACHM2, core group of **disease**-related genes; gene probes used for genetic profiling in healthcare screening and planning)

IT **Eye, disease**
(choroideremia, gene CHM, core group of **disease**-related genes; gene probes used for genetic profiling in healthcare screening and planning)

L148 ANSWER 20 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:567311 CAPLUS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: 125:189376
TITLE: Preparation of freeze-dry protein-degrading enzyme to be used in treating **contact lens**
INVENTOR(S): Nakayama, Hisayuki; Kimoto, Akihiro; Tsuchino, Noriko
PATENT ASSIGNEE(S): Senju Pharma Co, Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 08194192	A2	19960730	JP 1995-4400	19950113

PRIORITY APPLN. INFO.: JP 1995-4400 19950113
ED Entered STN: 24 Sep 1996
AB A method for freeze-drying a protein-degrading enzyme at -3.apprx.-40° in the presence of a surfactant followed by drying at <40° is described. The method improves the enzyme stability. Freeze-drying of Bioplase in the presence of lauroyl-L-triethanolamine glutamate and Polysorbate 80 was demonstrated. The preparation is suitable for cleaning contact lens.
IC ICM G02C007-04
ICS C12N009-50
ICA C11D003-386; C11D007-42; C11D017-00
CC 7-2 (Enzymes)
Section cross-reference(s): 63

ST freeze dry surfactant protein degrading enzyme; **contact lens** cleaning freeze dry enzyme

IT Surfactants
(preparation of freeze-dry protein-degrading enzyme to be used in treating **contact lens**)

IT Lenses
(contact, preparation of freeze-dry protein-degrading enzyme to be used in treating **contact lens**)

IT Enzymes
RL: PEP (Physical, engineering or chemical process); PROC (Process)
(protein-degrading, preparation of freeze-dry protein-degrading enzyme to be
used in treating **contact lens**)

IT 9014-01-1, Bioplase SP
RL: PEP (Physical, engineering or chemical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(freeze-drying of; preparation of freeze-dry protein-degrading enzyme to be used in treating **contact lens**)

IT 683-10-3
RL: NUU (Other use, unclassified); USES (Uses)
(preparation of freeze-dry protein-degrading enzyme to be used in treating **contact lens**)

IT 137-16-6, Lauroyl sarcosine sodium 9005-65-6, Polysorbate 80 53576-49-1
RL: NUU (Other use, unclassified); USES (Uses)
(surfactant; preparation of freeze-dry protein-degrading enzyme to be used in treating **contact lens**)

IT 9004-99-3
RL: PEP (Physical, engineering or chemical process); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(**surfactant**; preparation of freeze-dry **protein**-degrading enzyme to be used in treating **contact lens**)

L148 ANSWER 21 OF 45 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2005-589513 [60] WPIX
CROSS REFERENCE: 2001-381642 [40]
DOC. NO. CPI: C2005-177691
TITLE: Treating disorder or disease eg wound healing comprises use of multimeric polypeptide comprising **collectin** family scaffold linked to extracellular domain of tumor necrosis factor superfamily to form at least a dimer of trimer units.
DERWENT CLASS: B04 D16
INVENTOR(S): KORNBLUTH, R S
PATENT ASSIGNEE(S): (KORN-I) KORNBLUTH R S
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
US 2005158831	A1	20050721	(200560)*		33	C12P021-02

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2005158831	A1 Provisional	US 1998-111471P	19981209

Cont of US 1999-454223 19991209
US 2005-87348 20050322

PRIORITY APPLN. INFO: US 1998-111471P 19981209; US
1999-454223 19991209; US
2005-87348 20050322

INT. PATENT CLASSIF.:
MAIN: C12P021-02
SECONDARY: C07H021-04; C07K014-525; C12N001-21; C12N015-74

BASIC ABSTRACT:

US2005158831 A UPAB: 20050920

NOVELTY - Treating a disorder or disease in a subject comprises administering a multimeric polypeptide of trimer units, each of which comprising a **collectin** family scaffold operably linked to an extracellular domain of a tumor necrosis factor superfamily (TNFSF) polypeptide to form a polypeptide trimer, where the multimeric polypeptide is at least a dimer of trimer units.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:
(1) an isolated nucleic acid sequence encoding a multimeric polypeptide;
(2) an expression vector containing the nucleic acid sequence; (3) a host cell containing the expression vector; (4) a pharmaceutical composition comprising the nucleic acid sequence;
(5) a method for producing a multimeric polypeptide of trimer units comprises culturing the host cell under conditions to produce the multimeric polypeptide;
(6) a method of stimulating a biological response in a subject; (7) a method of stimulating angiogenesis in a subject; (8) a method of inhibiting angiogenesis in a subject; and (9) a method of promoting wound healing in a subject. ACTIVITY - Angiogenesis inhibitor; Angiogenesis stimulator; Vulnerary.; Cardiant; Vasotropic; Antiinflammatory; Immunosuppressive; Antimicrobial; Virucide; Cytostatic; **Ophthalmological.** No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The method is useful in treating a disorder or disease such as an inflammatory disorder, autoimmune disease, infectious disease, viral infectious disease or a tumor. The polypeptide is also useful in methods for stimulating a biological response in subjects with a tumor or HIV positive cells, inhibiting or stimulating angiogenesis in subjects with coronary artery disease, a tumor or retinal neovascularization and promoting wound healing in a subject with a poorly healing wound (all claimed).

Dwg.0/7

FILE SEGMENT: CPI
FIELD AVAILABILITY: AB; DCN
MANUAL CODES: CPI: B04-E02H; B04-E08; B04-F0100E; B04-N08; B14-A01; B14-A02; B14-C03; B14-F01E; B14-F02D; B14-F02F1; B14-F02F2; B14-G02D; B14-H01; **B14-N03**; B14-N17B; B14-S03A; D05-H12C; D05-H12E; D05-H14; D05-H17C

TECH UPTX: 20050920

TECHNOLOGY FOCUS - BIOTECHNOLOGY - Preferred Method: Treating a disorder or disease in a subject comprises:
(1) administering a multimeric polypeptide of trimer units, each of which comprising a **collectin** family scaffold operably linked to an extracellular domain of a tumor necrosis factor superfamily (TNFSF) polypeptide to form a polypeptide trimer, where the multimeric polypeptide is at least a dimer of trimer units; or
(2) a nucleic acid sequence encoding the multimeric polypeptide.
The disorder or disease is an inflammatory disorder, autoimmune disease, infectious disease, viral infectious disease or a tumor. The TNFSF polypeptide is TWEAK, VEGI or APRIL.

Stimulating a biological response in a subject comprises administering to the subject the composition comprising the multimeric polypeptide of trimer units or the polynucleotide encoding the multimeric polypeptide. The subject has tumor or HIV positive cells.
Stimulating angiogenesis in a subject comprises administering to the subject a composition comprising the multimeric polypeptide of trimer units or the polynucleotide encoding the multimeric polypeptide. The subject has coronary artery disease.
Inhibiting angiogenesis in a subject comprises administering to the subject a composition comprising the multimeric polypeptide of trimer units or the polynucleotide encoding the multimeric polypeptide. The subject has tumor or retinal neovascularization.
Promoting wound healing in a subject comprises administering a composition comprising the multimeric polypeptide of trimer units or a polynucleotide encoding the multimeric polypeptide. The subject has a poorly healing wound.
Preferred Host Cell: The host cell is a prokaryote.

L148 ANSWER 22 OF 45 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-093058 [08] WPIX
CROSS REFERENCE: 2003-229219 [22]; 2004-293804 [27]
DOC. NO. CPI: C2003-023304
TITLE: Pharmaceutical composition for treating asthma, has antisense oligonucleotide containing less percentage of adenosine, targeted to nucleic acids associated with lung airway or lung dysfunction, and bronchodilating agent.
DERWENT CLASS: B04 B05 C03 D16 D21
INVENTOR(S): AGUILAR, D; KATZ, E; LI, Y; MILLER, S; NYCE, J W; PABALAN, J; SANDRASAGRA, A; SHAHABUDDIN, S; TANG, L
PATENT ASSIGNEE(S): (EPIG-N) EPIGENESIS PHARM INC
COUNTRY COUNT: 100
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2002085309	A2	20021031	(200308)*	EN	763	A61K000-00
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW						
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW						
AU 2002305236	A1	20021105	(200433)			A61K000-00

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002085309	A2	WO 2002-US13143	20020423
AU 2002305236	A1	AU 2002-305236	20020423

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002305236	A1 Based on	WO 2002085309

PRIORITY APPLN. INFO: US 2001-286036P 20010424
INT. PATENT CLASSIF.:

MAIN: A61K000-00

BASIC ABSTRACT:

WO 200285309 A UPAB: 20040525

NOVELTY - A pharmaceutical composition (C1) comprises: (a) a first active agent (I), comprising oligonucleotide(s) (oligo(s)) containing about 1-15% of (A) and being anti-sense to a target nucleic acid; and

(b) a second active agent (II), comprises a bronchodilating agent.

DETAILED DESCRIPTION - A pharmaceutical composition (C1) comprises: (a) a first active agent (I), comprising oligonucleotide(s) (oligo(s)), effective for alleviating bronchoconstriction, respiratory tract inflammation, allergies, and reducing adenosine sensitivity, levels of adenosine (A) or (A) receptors, surfactant depletion or hyposecretion, when administered to a mammal. The oligo containing about 1-15% of (A) and being anti-sense to a target comprises: (i) the initiation codon,

(ii) the coding region,

(iii) the 5'-end or 3'-end genomic flanking regions, (iv) the 5' or 3' intron-exon junctions or regions within 2-10 nucleotides of the junctions of at least one gene encoding or regulating expression of a target polypeptide associated with lung airway or lung dysfunction or cancer, or is anti-sense to the corresponding mRNA; combinations, multiple target anti-sense oligos, or their mixtures; (b) a second active agent (II), comprises a bronchodilating agent; and

(c) a carrier or diluent.

An INDEPENDENT CLAIM is also included for a kit, that comprises: (a) a delivery device, in separate containers, (b) the oligo(s) of (C1), and (c) instructions for adding a carrier and for use of the kit. ACTIVITY - Antiallergic; Antiinflammatory; Antiasthmatic; Analgesic; Hypotensive; Immunosuppressive; Cytostatic.

MECHANISM OF ACTION - beta Adrenergic Agonist; Antisense therapy. Neonatal New Zealand white Pasteurella-free rabbits were immunized intraperitoneally within 24 hours of birth with 312 antigen units/ml house dustmite extract mixed with 10% kaolin. Immunizations were repeated weekly for the first month and then biweekly for the next 2 months. At 3-4 months of age, eight sensitized rabbits were anesthetized. The rabbits were then intubated with a 4.0 mm intratracheal tube. A polyethylene catheter of external diameter 2.4 mm with an attached latex balloon was passed into the esophagus and maintained at the same distance (approximately 16 cm) from the mouth throughout the experiments. The intratracheal tube was attached to a heated Fleisch pneumotachograph and flow was measured using a Validyne differential pressure transducer. The esophageal balloon was attached to one side of the differential pressure transducer, and the outflow of the intratracheal tube was connected to the opposite side of the pressure transducer to allow recording of transpulmonary pressure. Flow was integrated to give a continuous tidal volume, and measurements of total lung resistance (RL) and dynamic compliance (Cdyn) were calculated at isovolumetric and flow zero points, respectively. Animals were randomized and on Day 1 pretreatment values for PC50 were obtained for aerosolized adenosine. Anti-sense (HAdA1AS) (5'-GATGGAGGGCGGCATGGCGGG-3') or mismatched control (HAdA1MM) oligonucleotides (5'-GTAGCAGGCGGGGATGGGGGC- 3') were dissolved in sterile physiological saline at a concentration of 5000 micro g (5 mg) per 1.0 ml. Animals were subsequently administered the aerosolized anti-sense or mismatch oligonucleotide by the intratracheal tube twice daily for two days. Four randomly selected allergic rabbits were administered anti-sense oligonucleotide and four the mismatched control oligonucleotide. On the morning of the third day, PC50 values (the concentration of aerosolized adenosine in mg/ml required to reduce the dynamic compliance of the bronchial airway 50% from the baseline value) were obtained and compared to PC50 values obtained for these animals prior to exposure to oligonucleotide. The results showed that animals receiving the anti-sense oligonucleotide showed an order of magnitude increase in the dose of aerosolized adenosine required to reduce

dynamic compliance of the lung by 50%. No effect of the mismatched control oligonucleotide upon PC50 values was observed. No toxicity was observed in any animal receiving either anti-sense or control inhaled oligonucleotide.
USE - (I) is useful for preventing or treating a respiratory, lung, or malignant disease or condition which involves administering to a subject, preferably a human, simultaneously, sequentially, or separately administering (I) and (II). (I) is administered for alleviating bronchoconstriction or lung inflammation, or allergies, reducing (A) or (A) receptor levels, or (A) hypersensitivity, or surfactant depletion or hyposecretion. The administered composition comprises oligo(s) and is administered to reduce the production or availability, or to increase the degradation of the target mRNA or to reduce the amount of target polypeptide present in the lungs. The pulmonary obstruction, and/or bronchoconstriction and/or lung inflammation, allergies and/or surfactant hypoproduction are associated with a disease or condition such as pulmonary vasoconstriction, inflammation, allergies, asthma, impeded respiration, respiratory distress syndrome (RDS), pain, cystic fibrosis (CF), allergic rhinitis (AR), pulmonary hypertension, emphysema, chronic obstructive pulmonary disease (COPD), pulmonary transplantation rejection, pulmonary infections, bronchitis or cancer. The oligo is obtained by selecting fragments of a target nucleic acid having at least 4 contiguous nucleic acids chosen from G and C, obtaining a first oligonucleotide 4-60 nucleotides long which comprises the selected fragment and has a C and G nucleic acid content of up to and including about 15%, and obtaining a second oligonucleotide 4-60 nucleotides long comprising a sequence which is anti-sense to the selected fragment, the second oligonucleotide having an A base content up to and including about 15% (all claimed).
ADVANTAGE - The reduced adenosine content of the anti-sense oligos corresponding to thymidines present in the target RNA serves to prevent the breakdown of the oligos into products that free adenosine into the system e.g., lung, brain, heart, kidney, etc, tissue environment and thereby, to prevent any unwanted effects due to it. Dwg.0/4

FILE SEGMENT: CPI
FIELD AVAILABILITY: AB; GI; DCN
MANUAL CODES: CPI: B04-A06; B04-B03C; B04-E06; B04-E08; B06-D02; B07-D04D; B10-B02F; B10-B03B; B11-C04; B14-A01; B14-C01; B14-C03; B14-F02B; B14-F02D; B14-G02A; B14-G02C; B14-H01B; B14-J02D2; B14-K01A; B14-K01D; B14-K01F; B14-N04; B14-S03B; B14-S12; C04-A06; C04-B03C; C04-E06; C04-E08; C06-D02; C07-D04D; C10-B02F; C10-B03B; C11-C04; C14-A01; C14-C01; C14-C03; C14-F02B; C14-F02D; C14-G02A; C14-G02C; C14-H01B; C14-J02D2; C14-K01A; C14-K01D; C14-K01F; C14-N04; C14-S03B; C14-S12; D05-H12D2; D05-H12D4; D05-H12E; D08-A; D08-B

TECH UPTX: 20030204

TECHNOLOGY FOCUS - BIOTECHNOLOGY - Preferred Composition: In (I), the oligo is (A)-free. The oligo is antisense to above mentioned targets, where the target polypeptide(s) is associated with lung or airway dysfunction or cancer, comprises peptide factors or transmitters, antibodies, cytokines, chemokines, enzymes, binding proteins, adhesion molecules, their receptors, or malignancy associated proteins. Preferably, the polypeptides comprises transcription factors, stimulating or activating peptide factors, cytokines, cytokine receptors, chemokines, chemokine receptors, (A) receptors, bradykinin receptors, endogenously produced specific or non-specific enzymes, immunoglobulins, antibodies, antibody receptors, central nervous system (CNS) or peripheral nervous or non-nervous system receptors, CNS or peripheral nervous or non-nervous system peptide transmitters, adhesion molecules, defensins, growth factors, vasoactive peptides or receptors, binding proteins or malignancy associated proteins.

The encoded polypeptide targetted comprises e.g. (A) receptors A1, A2a, A2b, or A3, bradykinin receptors B1 or B2, NFkappaB transcription factor, endothelial leukocyte adhesion molecule (ELAM-1), monocyte activating factor.
Optionally, the encoded polypeptides comprises e.g. a H2A histone family member N, tubulin beta polypeptide, expressed sequence tags (ESTs) (AI095492), ESTs (AI138216), ESTs (AI128305), ESTs (AI125229), ESTs (AI041482), ESTs (AI051839).
One or more (A)s are substituted by a universal base comprising heteroaromatic base which bind to a thymidine base but have antagonist activity and less than about 0.3 of the (A) base agonist or antagonist activity at the adenosine A1, A2a, A2b or A3 receptors.
The heteroaromatic bases comprise pyrimidines or purines, which may be substituted by O, halo, NH2, SH, SO, SO2, SO3, COOH, branched or fused primary or secondary amino, alkyl, alkenyl, alkynyl, cycloalkyl, heterocycloalkyl, aryl, heteroaryl, alkoxy, alkenoxy, acyl, cycloacyl, arylacyl, alkynoxy, cycloalkoxy, aroyl arylthio, arylsulfoxyl, halocycloalkyl, alkylcycloalkyl, alkenylcycloalkyl, alkynylcycloalkyl, haloaryl, alkylaryl, alkynylaryl, alkynylaryl, arylalkyl, arylalkenyl, arylalkynyl, arylcycloalkyl, which may be further substituted by O, halo, NH2, primary, secondary or tertiary amine, SH, SO, SO2, SO3, cycloalkyl, heterocycloalkyl or heteroaryl. The purines are substituted at positions 1, 2, 3, 6, and/or 8, the pyrimidines are substituted at positions 2, 3, 4, 5 and/or 6, and the purines and pyrimidines have the chemical formula (F1) and (F2) respectively.
The universal base comprises 3-nitropyrrole-2'-deoxynucleoside, 5-nitro-indole, 2-deoxyribosyl-(5-nitroindole), 2-deoxyribofuranosyl-(5-nitroindole), 2'-deoxyinosine, 2'-deoxynebularine, 6H,8H-3,4-dihydropyrimido(4,5-c)oxazine-7-one or 2-amino-6-methoxyaminopurine.
In (I), one or more methylated cytosine(s) (mC) are substituted for a C in one or more CpG dinucleotide(s), if present in the oligo(s).
One or more mononucleotide(s) of (I) are linked or modified by one or more of methylphosphonate, 5'-N-carbamate, phosphotriester, phosphorothioate, phosphorodithioate, boranophosphate, formacetal, thioformacetal, thioether, carbonate, carbamate, sulfate, sulfonate, sulfamate, sulfonamide, sulfone, sulfite, sulfoxide, sulfide; hydroxylamine, methylene(methylimino) (MMI), methoxymethyl (MOM), methoxyethyl (MOE), methyleneoxy(methylimino) (MOMI), 2'-O-methyl, phosphoramidate, or C-5 substituted residues.
The mononucleotide residues are linked by phosphorothioate residues.
(I) comprises about 7 to about 60 mononucleotides.
The antisense oligo is operatively linked to, or complexed with, an agent chosen from cell internalized or up-taken agents and cell targeting agents.
The oligo is operatively linked to a vector.
(I) is present in an amount of about 0.01 to about 99.99 w/w of the composition, and the carrier comprises hydrophobic carrier such as lipid vesicles, optionally liposomes, or particles, optionally microcrystals. Preferably, the carrier comprises liposomes and the liposomes comprise the antisense oligo.
(C1) further comprises an agent chosen from other therapeutic agents, surfactants, flavoring and coloring agents, fillers, volatile oils, buffering agents, dispersants, RNA inactivating agents, anti-oxidants, flavoring agents, propellants or preservatives.
The other therapeutic or bioactive agents may be chosen from e.g. analgesics, pre-menstrual medications, menopausal agents, anti-aging agents, anti-anxiolytic agents, mood disorder agents, antidepressants, anti-bipolar mood agents.
The surfactant comprises e.g. surfactant protein A, surfactant protein B, surfactant protein C, **surfactant protein D,**

surfactant protein E, partially or fully saturated phosphatidyl choline (other than dipalmitoyl), dipalmitoylphosphotidylcholine, phosphotidylcholine, phosphotidylglycerol, phosphotidylinositol.
Preferably, (C1) comprises one or more oligo(s), a beta2 adrenergic agonist, a surfactant, and a carrier or diluent for the oligo, the beta2 adrenergic agent and the surfactant.
The agent is an RNA inactivating agent that comprises an enzyme, optionally a ribozyme.
(C1) is systemic or topical formulation comprising an oral, intrabuccal, intrapulmonary, rectal, intrauterine, intratumor, intracranial, nasal, intramuscular, subcutaneous, intravascular, intrathecal, inhalable, transdermal, intracavitary, implantable, iontophoretic, **ocular**, vaginal, intrarticular, intravenous, intramuscular, intraglandular, intraorgan, intralymphatic, slow release or enteric coating formulation.
(C1) is preferably provided in an implant, a capsule or cartridge.
Most preferably, (C1) is a nasal, intrapulmonary, respirable, or inhalable, aerosol formulation of particle size 0.5-10 micron or about 8-100 micron. (C1) is in single or multiple unit form, or is in bulk.
R1, R2 = H, alkyl, alkenyl or alkynyl;
R3 = H, aryl, dicycloalkyl, dicycloalkenyl, dicycloalkynyl, cycloalkyl, cycloalkenyl, cycloalkynyl, O-cycloalkyl, O-cycloalkenyl, O-cycloalkynyl, NH2-alkylamino-ketoxyalkyloxy-aryl, mono-alkylaminoalkyl-N-alkylamino-SO2aryl, or dialkylaminoalkyl-N-alkylamino-SO2aryl;
R4, R5 = R1;
R4 and R5 together = R3; and
the pyrimidines and purines optionally are theophylline, caffeine, dyphylline, etophylline, acephylline, piperazine, bamifylline, enprofylline or xanthine.
TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Components: (II) comprises a beta2 adrenergic agonist, anti-cholinergic agent, theophylline, anti-histaminic agent, (A) receptor antagonist or glucocorticosteroid.
The beta adrenergic agonist is chosen from ephedrine, isoproterenol, isoetharine, epinephrine, metaproterenol, terbutaline, fenoterol, procaterol, albuterol, salbutamol, pirbuterol, formoterol, biloterol, bambuterol, salmeterol or seretide.

L148 ANSWER 23 OF 45 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 1999-478910 [40] WPIX
CROSS REFERENCE: 2002-033698 [04]; 2003-102285 [09]
DOC. NO. CPI: C1999-140868
TITLE: Fusion polypeptide comprising an antigen and an APC binding domain of an opsonin, used to treat, e.g. systemic lupus erythematosis.
DERWENT CLASS: B04 D16
INVENTOR(S): SEGAL, A H; SEGAL, A
PATENT ASSIGNEE(S): (GENI-N) GENITRIX LLC; (GENI-N) GENITRIX LTD
COUNTRY COUNT: 84
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 9936507	A1	19990722	(199940)*	EN	102	C12N005-10
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW						
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW						
AU 9922301	A	19990802	(199954)			C12N005-10
US 6224870	B1	20010501	(200126)			A61K039-00

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9936507	A1	WO 1999-US894	19990115
AU 9922301	A	AU 1999-22301	19990115
US 6224870	B1 CIP of	US 1997-788143	19970124
		US 1998-7711	19980115

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 9922301	A Based on	WO 9936507

PRIORITY APPLN. INFO: US 1998-7711 19980115; US 1997-788143 19970124

INT. PATENT CLASSIF.:

MAIN: A61K039-00; C12N005-10

SECONDARY: A01N043-04; A61K031-70; A61K039-385; A61K039-395; C07K016-28; C07K019-00; C12N015-13; C12N015-62; C12N015-86; C12P021-08

BASIC ABSTRACT:

WO 9936507 A UPAB: 20030206

NOVELTY - Immune responses are modulated using a fusion polypeptide (FP) comprising an antigen (Ag) and an Ag presenting cell (APC) binding domain (bd) of an opsonin (OP), or a nucleic acid (NA) encoding the FP.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) modulating an immune response to an Ag by administering a NA molecule (NAM) encoding a FP comprising an Ag and an APC bd of an OP; (2) a method similar to (1) comprising administering a NAM encoding FP comprising an Ag and OP;

(3) a method similar to (1) comprising administering a NAM encoding FP comprising an Ag and a first portion of an OP which, when associated with a second portion of the OP, forms an APC bd or a multichain polypeptide complex; (4) a method similar to (1) comprising administering a NAM encoding FP comprising a secretory signal sequence (SSS), an Ag, a first cell bd of a ligand for a cell surface polypeptide (CSP) and a second cell bd of a ligand for a CSP;

(5) a method similar to (1) comprising administering a NAM encoding FP comprising an Ag, an OP and a cell bd of a ligand for a CSP; (6) isolated NA comprising a nucleotide (nt) sequence encoding FP comprising an Ag and an APC bd of an OP, where the OP is selected from: c-reactive protein (crp), complement component C1q and cc C3, complement fragment C3b and C4b, a **collectin**, mannose binding protein, conglutinin and surfactant protein A or D; (7) an isolated NA comprising a nt sequence encoding a FP comprising an Ag and an APC bd of alpha-2-macroglobulin, where the Ag is not: (i) a portion of the adenovirus fiber protein; (ii) carbonic anhydrase, or (iii) a heptapeptide which comprises a cleavage site for the TEV protease; (8) a polypeptide encoded by the NA as in (6) and (7); (9) a vector containing the NA as in (6) or (7); (10) a host cell transfected with the vector of (9); (11) a multichain polypeptide complex comprising FP comprising an Ag and a first portion of an OP which, when associated with a second portion of OP, forms an APC bd, covalently associated with the second portion, and (12) a composition comprising the FP as in (8), or the multichain polypeptide complex of (11), admixed with APCs. ACTIVITY - Immunomodulatory; Antitumor; Antiinflammatory; Antiallergic.

MECHANISM OF ACTION - None given.

USE - The NAMs or FP's are useful as immunogens for vaccination against a variety of diseases such as diabetes mellitus, arthritis, multiple sclerosis, myasthenia gravis, systemic lupus erythematosis, autoimmune thyroiditis, dermatitis, psoriasis, Sjoegren's Syndrome, alopecia areata, Crohn's disease, aphthous ulcer, conjunctivitis, asthma, scleroderma, vaginitis, proctitis, drug eruptions, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, acute necrotizing hemorrhagic encephalopathy, idiopathic bilateral progressive sensorineural hearing loss, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Stevens Johnson syndrome, idiopathic sprue, lichen planus, sarcoidosis, primary biliary cirrhosis, uveitis posterior and interstitial lung fibrosis.
ADVANTAGE - The fusion polypeptides improve uptake of antigen by APCs.
Dwg.0/0

FILE SEGMENT: CPI
FIELD AVAILABILITY: AB; DCN
MANUAL CODES: CPI: B04-B04C1; B04-C01G; B04-E02; B04-E08; B04-F0100E; B04-H20; B04-H20A; B04-N03; B14-C03; B14-E10C; B14-F03; B14-G02; B14-G02A; B14-G03; B14-H01; B14-K01; **B14-N03**; B14-N17; B14-S01; B14-S11; D05-H07; D05-H12A; D05-H12C; D05-H12E; D05-H14; D05-H17C

TECH UPTX: 19991004
TECHNOLOGY FOCUS - BIOTECHNOLOGY - Preferred Opsonin: The opsonin is fibronectin, alpha-2-macroglobulin, c-reactive protein (crp), complement component C1q and C3, complement fragment C3b and C4b, a **collectin** , mannose binding protein, conglutinin and surfactant protein A or D. The antigen is an antigen of a bacterium, virus, fungus or parasite, or is an antigen involved in autoimmune disease, allergy or graft rejection and it is especially a tumor antigen.
Preparation: The fusion polypeptides may be prepared using known methods.

L148 ANSWER 24 OF 45 EMBASE

ACCESSION NUMBER: 2006254237 EMBASE Full-text<<LOGINID::20061004>>
TITLE: Solid-phase chemical tools for glycobiology.
AUTHOR: Larsen K.; Thygesen M.B.; Guillaumie F.; Willats W.G.T.; Jensen K.J.
CORPORATE SOURCE: K.J. Jensen, Department of Natural Sciences, Section for Bioorganic Chemistry, Royal Veterinary and Agricultural University, DK-1871 Frederiksberg, Denmark. kjj@kvl.dk
SOURCE: Carbohydrate Research, (24 Jul 2006) Vol. 341, No. 10, pp. 1209-1234. .
Refs: 153
ISSN: 0008-6215 CODEN: CRBRAT
PUBLISHER IDENT.: S 0008-6215(06)00234-5
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Jul 2006
Last Updated on STN: 10 Jul 2006

ABSTRACT: Techniques involving solid supports have played crucial roles in the development of genomics, proteomics, and in molecular biology in general. Simularly, methods for immobilization or attachment to surfaces and resins have become ubiquitous in sequencing, synthesis, analysis, and screening of

oligonucleotides, peptides, and proteins. However, solid-phase tools have been employed to a much lesser extent in glycobiology and glycomics. This review provides a comprehensive overview of solid-phase chemical tools for glycobiology including methodologies and applications. We provide a broad perspective of different approaches, including some well-established ones, such as immobilization in microtiter plates and to cross-linked polymers. Emerging areas such as glycan microarrays and glycan sequencing, quantum dots, and gold nanoparticles for nanobioscience applications are also discussed. The applications reviewed here include enzymology, immunology, elucidation of biosynthesis, and systems biology, as well as first steps toward solid-supported sequencing. From these methods and applications emerge a general vision for the use of solid-phase chemical tools in glycobiology.

CONTROLLED TERM: Medical Descriptors:
*glycobiology
solid
methodology
microtiter plate assay
cross linking
microarray analysis
quantum dot
nanoparticle
enzymology
immunology
biosynthesis
adsorption
enzyme linked immunosorbent assay
amination
protein carbohydrate interaction
high performance liquid chromatography
Pseudomonas aeruginosa
enzyme activity
Viscum album
catalysis
enzyme specificity
protein binding
mass spectrometry
matrix assisted laser desorption ionization time of flight mass spectrometry
lectin binding
aqueous solution
glycosylation
crystal structure
gene mapping
imaging
Caenorhabditis elegans
rabbit
rat strain
affinity chromatography
drug purification
nucleotide sequence
amino acid sequence
nonhuman
short survey
priority journal

CONTROLLED TERM: Drug Descriptors:
*carbohydrate
polymer
glycan

gold
oligosaccharide
aglycone
lectin
glycoprotein
monosaccharide
bovine serum albumin
proteoglycan
glycolipid
pyroxylin
polystyrene
water
peptide derivative
glycopeptide
sepharose
glycosaminoglycan
RANTES
chymotrypsin
edetic acid
surfactant protein D: DV, drug development
antiinfective agent: DV, drug development

CAS REGISTRY NO.: (gold) 7440-57-5; (pyroxylin) 9004-70-0; (polystyrene) 9003-53-6; (water) 7732-18-5; (sepharose) 61970-08-9; (chymotrypsin) 9004-07-3, 9014-64-6; (edetic acid) 150-43-6, 60-00-4

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ACCESSION NUMBER: 2005365215 EMBASE Full-text<<LOGINID::20061004>>
TITLE: Surfactant and lung inflammation.
AUTHOR: Reid K.B.M.; Clark H.; Palaniyar N.
CORPORATE SOURCE: Prof. K.B.M. Reid, MRC Immunochemistry Unit, Department of Biochemistry, University of Oxford, South Parks Road, Oxford OX1 ODQ, United Kingdom. kenneth.reid@bioch.ox.ac.uk
SOURCE: Thorax, (2005) Vol. 60, No. 8, pp. 620-622.
Refs: 40
ISSN: 0040-6376 CODEN: THORA7
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 004 Microbiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Oct 2005
Last Updated on STN: 27 Oct 2005
CONTROLLED TERM: Medical Descriptors:
*pneumonia: DT, drug therapy
*pneumonia: ET, etiology
epithelium cell
respiratory distress syndrome: DT, drug therapy
adult respiratory distress syndrome: DT, drug therapy
respiratory failure: DT, drug therapy
respiratory failure: ET, etiology
immunogenicity
bacterial cell wall
protein binding
DNA binding
lung emphysema
cystic fibrosis

Pseudomonas aeruginosa
human
nonhuman
clinical trial
editorial
priority journal
Drug Descriptors:
*surfactant: CT, clinical trial
*surfactant: CB, drug combination
*surfactant: DT, drug therapy
surfactant protein A: CB, drug combination
surfactant protein A: DT, drug therapy
surfactant protein D: CB, drug combination
surfactant protein D: DT, drug therapy

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ACCESSION NUMBER: 2004463722 EMBASE Full-text<<LOGINID::20061004>>

TITLE: Anti-Aspergillus fumigatus efficacy of pentraxin 3 alone and in combination with antifungals.

AUTHOR: Gaziano R.; Bozza S.; Bellocchio S.; Perruccio K.; Montagnoli C.; Pitzurra L.; Salvatori G.; De Santis R.; Carminati P.; Mantovani A.; Romani L.

CORPORATE SOURCE: L. Romani, Dept. of Exp. Med. and Biochem. Sci., Microbiology Section, University of Perugia, Via del Giochetto, 06122 Perugia, Italy. lromani@unipg.it

SOURCE: Antimicrobial Agents and Chemotherapy, (2004) Vol. 48, No. 11, pp. 4414-4421. .
Refs: 47
ISSN: 0066-4804 CODEN: AMACCQ

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 2 Dec 2004
Last Updated on STN: 2 Dec 2004

ABSTRACT: The collectin pentraxin 3 (PTX3) is an essential component of host resistance to pulmonary aspergillosis. Here we examined the protective elects of administration of PTX3 alone or together with deoxycholate amphotericin B (Fungizone) or liposomal amphotericin B (AmBisome) against invasive aspergillosis in a murine model of allogeneic bone marrow transplantation. PTX3, alone or in combination with the polyenes, was given intranasally or parenterally either before, in concomitance with, or after the intranasal infection with Aspergillus fumigatus conidia. Mice were monitored for resistance to infection and parameters of innate and adaptive T-helper immunity. The results showed the following: (i) complete resistance to infection and reinfection was observed in mice treated with PTX3 alone; (ii) the protective effect of PTX3 was similar or superior to that observed with liposomal amphotericin B or deoxycholate amphotericin B, respectively; (iii) protection was associated with accelerated recovery of lung phagocytic cells and T-helper-1 lymphocytes and concomitant decrease of inflammatory pathology; and (iv) PTX3 potentiated the therapeutic efficacy of suboptimal doses of either antimycotic drug. Together, these data suggest the potential therapeutic use of PTX3 either alone or as an adjunctive therapy in A. fumigatus infections.

CONTROLLED TERM: Medical Descriptors:
*Aspergillus fumigatus
drug efficacy
antifungal activity
host resistance
lung aspergillosis: DT, drug therapy
allogenic bone marrow transplantation
conidium
helper cell
immunity
infection resistance
reinfection
phagocyte
drug potentiation
nonhuman
female
mouse
animal experiment
animal model
article
priority journal
Drug Descriptors:
*antifungal agent: CB, drug combination
*antifungal agent: CM, drug comparison
*antifungal agent: DT, drug therapy
*pentraxin: CB, drug combination
*pentraxin: CM, drug comparison
*pentraxin: DT, drug therapy
*pentraxin: NA, intranasal drug administration
*pentraxin: IP, intraperitoneal drug administration
*pentraxin: PA, parenteral drug administration
*pentraxin 3: CB, drug combination
*pentraxin 3: CM, drug comparison
*pentraxin 3: DT, drug therapy
*pentraxin 3: NA, intranasal drug administration
*pentraxin 3: IP, intraperitoneal drug administration
*pentraxin 3: PA, parenteral drug administration
collectin: CB, drug combination
collectin: CM, drug comparison
collectin: DT, drug therapy
collectin: NA, intranasal drug administration
collectin: IP, intraperitoneal drug administration
collectin: PA, parenteral drug administration
amphotericin B deoxycholate: CB, drug combination
amphotericin B deoxycholate: CM, drug comparison
amphotericin B deoxycholate: DT, drug therapy
amphotericin B deoxycholate: IP, intraperitoneal drug administration
amphotericin B lipid complex: CB, drug combination
amphotericin B lipid complex: CM, drug comparison
amphotericin B lipid complex: DT, drug therapy
amphotericin B lipid complex: IP, intraperitoneal drug administration
unclassified drug
CAS REGISTRY NO.: (collectin) 260234-88-6
CHEMICAL NAME: (1) Fungizone; (2) Ambisome
COMPANY NAME: (1) Bristol Myers Squibb (Italy); (2) Gilead (Italy); Sigma Tau (Italy)

L148 ANSWER 27 OF 45 JICST-EPlus COPYRIGHT 2006 JST on STN DUPLICATE 12

ACCESSION NUMBER: 1010938488 JICST-EPlus Full-text<<LOGINID::20061004>>
TITLE: **Surfactant Protein D (**
SP-D) and Systemic Scleroderma (SSc).
AUTHOR: MAEDA M
ICHIKI Y; AOYAMA Y; KITAJIMA Y
CORPORATE SOURCE: Gifu Prefectural Hospital, Gifu, Jpn
Gifu Univ., Gifu, Jpn
SOURCE: J Dermatol, (2001) vol. 28, no. 9, pp. 467-474. Journal Code: Z0757A (Fig. 6, Ref. 21)
ISSN: 0385-2407
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: English
STATUS: New
ABSTRACT:
We measured serum levels of **SP-D** in collagen diseases (110 cases) such as systemic scleroderma (SSc), scleroderma spectrum disorders (SSD), systemic lupus erythematodes (SLE), Sjoegren syndrome (Sjs), dermatomyositis (DM), rheumatoid arthritis (RA), and dermatitis (DE) (109 cases) as a control. Additionally, we performad a correlation analysis to determine how these levels were related to pulmonary fibrosis and function test (vitalcapacity, %DLco). The serum levels of **SP-D** increased in SSc patients with Barnett type III more than in SSc patients with Barnett type I or II, while they increased slightly in SSD (incomplete type of SSc) patients. The differences in these figures were statistically significant between the SSc (SSc & SSD) and non-SSc (SLE, DM, Sjs & RA) groups ($p<0.005$). The serum levels of **SP-D** in SSc patients with anti-topoisomerase I antibodies were statistically higher than those in SSc patients with other types of anti-nuclear antibodies. There was a statistically significant correlation between the severity of pulmonary fibrosis and the serum levels of **SP-D**, and a statistically negative correlation between **SP-D** levels and vital capacity or %DLco, but there was no proportional correlation with the forced expiatory volume (FEV10%). There was no statistical relationship between pre-and post-therapy with photopheresis; however, there was a statistical correlation between the serum levels of **SP-D** and KL-6. In the group of collagen diseases, plasma levels of **SP-D** were higher than serum levels of **SP-D**. Patients with SSc possess higher levels of **SP-D** than do those with other collagen diseases and dermatitis, which may correspond to the severity of pulmonary fibrosis. (author abst.)

CLASSIFICATION: GD07010X (616-079+)
CONTROLLED TERM: progressive systemic sclerosis; pulmonary surfactant; serum concentration; pulmonary fibrosis; systemic lupus erythematosus; Sjoegren syndrome; rheumatoid arthritis; dermatomyositis; pathophysiology; human(primates)
BROADER TERM: scleroderma; connective tissue disease; disease; skin disease; collagen disease; bioactive factor; factor; blood concentration; concentration(ratio); degree; fibrous disease; lung disease; respiratory tract disease; erythematosus; autoimmune disease; immunologic disease; xerostomia; salivary gland disease; mouth disease; stomatognathic disease; arthritis; inflammation; joint disease; bone and joint disease; rheumatism; lacrimal apparatus disease; eye disease; polymyositis; myositis; muscular disease; multiple disease

L148 ANSWER 28 OF 45 JICST-EPlus COPYRIGHT 2006 JST on STN
ACCESSION NUMBER: 1030193085 JICST-EPlus Full-text<<LOGINID::20061004>>

TITLE: Clinical Significance of Serum KL-6 and SP-D for the Diagnosis and Treatment of Interstitial Lung disease in Patients with Diffuse Connective Tissue Disorders.

AUTHOR: OGAWA NORIYOSHI; SHIMOYAMA KUMIKO; KAWABATA HIROSHI; MASAKI YASUFUMI; WANO YUJI; SUGAI SUSUMU

CORPORATE SOURCE: Kanazawaidai Ketsuekimen'ekinaika

SOURCE: Riumachi (Official Journal of Japan College of Rheumatology), (2003) vol. 43, no. 1, pp. 19-28. Journal Code: Z0690A (Fig. 6, Tbl. 2, Ref. 19)
ISSN: 0300-9157

PUB. COUNTRY: Japan

DOCUMENT TYPE: Journal; Article

LANGUAGE: Japanese

STATUS: New

ABSTRACT:
Objective: To elucidate the clinical significance of serum KL-6 and SP-D for the diagnosis and treatment of interstitial lung disease in connective tissue disorders. Methods: 139 patients with various connective tissue disorders were subjected for the study, which included 46 cases of rheumatoid arthritis, 43 cases of Sjoegren's syndrome, 16 cases of SLE, 10 cases of systemic sclerosis, 9 cases of polymyositis/dermatomyositis, 6 cases of vasculitis syndrome, 5 cases of Behcet's disease and 4 cases of MCTD. Serum levels of KL-6 and SP-D were determined by enzyme-immunoassay. The sensitivity, specificity and accuracy of serum KL-6 and ***SP*** -D for the diagnosis of interstitial lung disease were compared with serum LDH. The relationship of serum KL-6 and SP-***D*** levels with high resolution CT (HRCT) of the lung and Gallium scintigraphy findings was analyzed. In some cases, serum levels of the two markers were determined monthly in the course of the disease. Results: When the serum levels of KL-6 and SP-D were measured simultaneously, the sensitivity to diagnose interstitial lung disease was 67.7%, the specificity was 98.1%, and the accuracy was 91.4%, while those of serum LDH were 45.2%, 88.9%, 79.1% respectively. In the patients with interstitial lung disease, those who had elevated serum levels of both KL-6 and SP-***D*** showed parenchymal collapse opacity-dominant pattern in HRCT. On the other hand, the patients with interstitial lung disease who had normal levels of serum KL-6 and SP-D or had elevation either in KL-6 or ***SP*** -D levels showed ground glass opacity-dominant pattern in HRCT. There was no significant correlation between serum marker levels and Gallium scintigraphy findings. When serum KL-6 and SP-D were measured monthly, the levels of both markers changed more specifically and sensitively to the lung disease activity compared with serum LDH.... (author abst.)

CLASSIFICATION: GI02000S (616.2-07)

CONTROLLED TERM: interstitial pneumonia; Sjoegren syndrome; pulmonary surfactant; membrane glycoprotein; serum concentration; complication; human(primates); X-ray computed tomography; systemic lupus erythematosus; progressive systemic sclerosis; dermatomyositis; mixed connective tissue disease; respirography; scintigraphy

BROADER TERM: pneumonia; inflammation; disease; lung disease; respiratory tract disease; obstructive lung disease; xerostomia; salivary gland disease; mouth disease; stomatognathic disease; rheumatoid arthritis; arthritis; joint disease; bone and joint disease; collagen disease; connective tissue disease; autoimmune disease; immunologic disease; rheumatism; lacrimal apparatus disease; eye disease; bioactive factor; factor; glycoprotein; protein;

membrane protein; blood concentration; concentration(ratio); degree; X-ray inspection; radiographic inspection; nondestructive inspection; inspection; computed tomography; diagnostic imaging; diagnosis; tomography; image technology; technology; radiography; erythematosus; skin disease; scleroderma; polymyositis; myositis; muscular disease; multiple disease; respiratory diagnosis; radioisotope diagnosis

SUPPLEMENTARY TERM: KL-6; **SP-D**

L148 ANSWER 29 OF 45 CABA COPYRIGHT 2006 CABI on STN DUPLICATE 11

ACCESSION NUMBER: 2003:158182 CABA Full-text<<LOGINID::20061004>>

DOCUMENT NUMBER: 20033130045

TITLE: Mannose-binding lectin deficiencies in infectious and inflammatory disorders

AUTHOR: Guardia, A.; Lozano, F.

CORPORATE SOURCE: Servei d'Immunologia, Institut Clinic d'Infeccions i Immunologia (ICII), Institut d'Investigacions Biomediques August Pi i Sunyer (IDIBAPS), Hospital Clinic, Villaroel 170, 08036 Barcelona, Spain. lozano@medicina.ub.es

SOURCE: Reviews in Medical Microbiology, (2003) Vol. 14, No. 2, pp. 41-52. 95 ref.
Publisher: Lippincott Williams & Wilkins. London
ISSN: 0954-139X

PUB. COUNTRY: United Kingdom

DOCUMENT TYPE: Journal

LANGUAGE: English

ENTRY DATE: Entered STN: 3 Oct 2003
Last Updated on STN: 3 Oct 2003

ABSTRACT:
Mannose-binding lectin (MBL) is a plasma protein belonging to the family of ***collectins*** , which are carbohydrate binding proteins (lectins) composed of a carbohydrate recognition domain and a collagen-like stalk domain. The MBL subunits associate to form multimeric complexes (resembling a bouquet of tulips) capable of recognizing carbohydrate patterns displayed at high density on bacteria, viruses, fungi and protozoa but not mammalian cells. Upon binding, MBL activates the complement system leading to opsonization or direct killing of microorganisms. The key role played by MBL in innate immune defence has led to the notion that MBL functions as an ante-antibody, which controls microbial invasion in the lag phase between infection and the generation of specific antibodies and cellular immunity. Surprisingly, a relatively large proportion of individuals (20%-40%) in all the human populations studied carry either homo- or heterozygous mutations in the MBL2 gene, which results in low plasma levels of MBL. This MBL deficiency is not normally associated with disease in healthy individuals. It may result, however, in increased susceptibility to infections in immunocompromised individuals and in premature and young children. This notion has been confirmed by recent published works, as reviewed in the present report. The effect of MBL deficiency in several infectious (i.e. meningococcal and streptococcal diseases, hepatitis B and C, HIV-1 infections, mycobacterial diseases, leishmaniasis, malaria, aspergillosis) and inflammatory diseases (i.e. systemic lupus erythematosus, rheumatoid arthritis, ***Sjogren*** 's syndrome, dermatomyositis, inflammatory bowel diseases, primary biliary cirrhosis) is discussed. These data indicate that MBL deficiency is not only a susceptibility marker but also a disease modifier of a large number of infectious and inflammatory processes, including autoimmune diseases.

CLASSIFICATION: VV055 Human Immunology and Allergology (New March 2000); VV080 Human Genetics and Molecular Medicine

(New June 2002); VV210 Prion, Viral, Bacterial and Fungal Pathogens of Humans (New March 2000); VV220 Protozoan, Helminth and Arthropod Parasites of Humans (New March 2000); VV600 Non-communicable Human Diseases and Injuries; ZZ360 General Molecular Biology (Discontinued March 2000)

SEQUENCE CODE: 1T; 2T; 0Y; 0L; CA; HE; PA

BROADER TERM: Aspergillus; Deuteromycotina; Eumycota; fungi; Hepadnaviridae; viruses; hepatitis C virus group; Flaviviridae; human immunodeficiency virus; Lentivirus; Retroviridae; Trypanosomatidae; Kinetoplastida; Sarcomastigophora; Protozoa; invertebrates; animals; Homo; Hominidae; Primates; mammals; vertebrates; Chordata; Mycobacteriaceae; Firmicutes; bacteria; prokaryotes; Neisseria; Neisseriaceae; Gracilicutes; Plasmodium; Plasmodiidae; Haemospororida; Apicomplexa; Streptococcus; Streptococcaceae

CONTROLLED TERM: aspergillosis; atherosclerosis; autoimmune diseases; bacterial diseases; binding proteins; blood disorders; cirrhosis; complement activation; Crohn's disease; cystic fibrosis; genetic polymorphism; heart diseases; hepatitis B; hepatitis C; HIV-1 infections; human diseases; immune system; immunological deficiency; lectins; leishmaniasis; malaria; molecular biology; molecular genetics; mutations; mycobacterial diseases; neoplasms; opsonins; phagocytosis; reviews; rheumatoid arthritis; **Sjogren**'s syndrome; spontaneous abortion; susceptibility; systemic lupus erythematosus; ulcerative colitis

SUPPLEMENTARY TERM: dermatomyositis; mannose-binding lectin; sarcoidosis

ORGANISM NAME: Aspergillus fumigatus; hepatitis B virus; hepatitis C virus; human immunodeficiency virus type 1; Leishmania; man; Mycobacterium; Neisseria meningitidis; Plasmodium falciparum; Streptococcus pneumoniae

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ACCESSION NUMBER: 2005:240400 BIOSIS Full-text<<LOGINID::20061004>>

DOCUMENT NUMBER: PREV200510029165

TITLE: **Surfactant protein D** is present in human **tear** fluid and the cornea and inhibits epithelial cell invasion by Pseudomonas aeruginosa.

AUTHOR(S): Ni, Minjian; Evans, David J.; Hawgood, Samuel; Anders, E. Margot; Sack, Robert A.; Fleiszig, Suzanne M. J. [Reprint Author]

CORPORATE SOURCE: Univ Calif Berkeley, Sch Optometry, Berkeley, CA 94720 USA fleiszig@socrates.berkeley.edu

SOURCE: Infection and Immunity, (APR 2005) Vol. 73, No. 4, pp. 2147-2156.
CODEN: INFIBR. ISSN: 0019-9567.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 29 Jun 2005
Last Updated on STN: 29 Jun 2005

ABSTRACT:We have previously shown that human **tear** fluid protects corneal epithelial cells against Pseudomonas aeruginosa in vitro and in vivo

and that protection does not depend upon **tear** bacteriostatic activity. We sought to identify the responsible **tear** component(s). The hypothesis tested was that **collectins** (collagenous calcium-dependent lectins) were involved. Reflex **tear** fluid was collected from healthy human subjects and examined for **collectin** content by enzyme-linked immunosorbent assay (ELISA) and Western blot with antibody against **surfactant protein D (SP -D)**, SP-A, or mannose-binding lectin (MBL). **SP-D**, but not SP-A or MBL, was detected by ELISA of human reflex **tear** fluid. Western blot analysis of whole **tears** and of high-performance liquid chromatography **tear** fractions confirmed the presence of ***SP*** -D, most of which eluted in the same fraction as immunoglobulin A. **SP-D tear** concentrations were calculated at similar to 2 to 5 mu g/ml. Depletion of **SP-D** with mannan-conjugated Sepharose or anti-**SP-D** antibody reduced the protective effect of **tears** against P. aeruginosa invasion. Recombinant human or mouse **SP-D** used alone reduced P. aeruginosa invasion of epithelial cells without detectable bacteriostatic activity or bacterial aggregation. Immunofluorescence microscopy revealed **SP-D** antibody labeling throughout the corneal epithelium of normal, but not gene-targeted **SP-D** knockout mice. **SP-D** was also detected in vitro in cultured human and mouse corneal epithelial cells. In conclusion, **SP-** ***D*** is present in human **tear** fluid and in human and mouse corneal epithelia. **SP-D** is involved in human **tear** fluid protection against P. aeruginosa invasion. Whether **SP-** ***D*** plays other roles in the regulation of other innate or adaptive immune responses at the **ocular** surface, as it does in the airways, remains to be explored.

CONCEPT CODE: Biochemistry studies - General 10060
Biochemistry studies - Proteins, peptides and amino acids 10064
Sense organs - Physiology and biochemistry 20004
Physiology and biochemistry of bacteria 31000

INDEX TERMS: Major Concepts
Biochemistry and Molecular Biophysics; Infection; Sense Organs (Sensory Reception)

INDEX TERMS: Parts, Structures, & Systems of Organisms
cornea: sensory system; corneal epithelium: sensory system; **tear** fluid: sensory system

INDEX TERMS: Chemicals & Biochemicals
mannose-binding lectin; **surfactant protein D; collectins;** mannan-conjugated Sepharose

INDEX TERMS: Methods & Equipment
enzyme-linked immunosorbent assay [ELISA]: laboratory techniques, immunologic techniques; high performance liquid chromatography: laboratory techniques, chromatographic techniques; Western blot: electrophoretic techniques, immunologic techniques, laboratory techniques

INDEX TERMS: Miscellaneous Descriptors
bacteriostatic activity

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human (common): host
Taxa Notes

Animals, Chordates, Humans, Mammals, Primates, Vertebrate

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
mouse (common): host
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrate

ORGANISM: Classifier
Pseudomonadaceae 06508
Super Taxa
Gram-Negative Aerobic Rods and Cocci; Eubacteria; Bacteria; Microorganisms
Organism Name
Pseudomonas aeruginosa (species): pathogen
Taxa Notes
Bacteria, Eubacteria, Microorganism

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ACCESSION NUMBER: 2005:302820 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV200510086478
TITLE: Rat strain differences in sleep after acute mild stressors and short-term sleep loss.
AUTHOR(S): Tang, Xiangdong; Liu, Xianling; Yang, Linghui; Sanford, Larry D. [Reprint Author]
CORPORATE SOURCE: Eastern Virginia Med Sch, Sleep Res Lab, Dept Pathol and Anat, POB 1980,700 Olney Rd, Norfolk, VA 23501 USA sanforld@evms.edu
SOURCE: Behavioural Brain Research, (MAY 7 2005) Vol. 160, No. 1, pp. 60-71.
CODEN: BBREDI. ISSN: 0166-4328.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Aug 2005
Last Updated on STN: 15 Aug 2005

ABSTRACT:Genetic and physiological diversity amongst rodent strains provide the potential for developing models that may give insight into factors that regulate sleep in response to environmental challenges. We examined home cage activity, behavioral performance in the open field and sleep after a number of mild stressors (cage change [CC], open field [OF]) and after I and 4 h of sleep deprivation (1hSD and 4hSD) in rat strains (Fischer 344 [F344], Lewis [LEW], Wistar [WST] and Sprague-Dawley [Sp-D], n = 16 per strain) that differ in behavior and sleep. F344 and WST rats had greater home cage locomotion than LEW and Sp-D rats, but F344 rats exhibited the least relative locomotion in OF. In 24 h baseline recordings of sleep, strain rankings were LEW = WST = Sp-D > F344 in rapid ***eye*** movement sleep (REM), and LEW = Sp-D > F344 and LEW > WST in non-REM (NREM). Compared to baseline, total sleep was reduced in all four strains after CC, OF and 1hSD, but not after 4hSD, in the first hour after treatment. Afterwards, increases in REM and NREM were seen after all treatments with the amount and time course varying across treatments and strains. CC induced the weakest and 4hSD the largest effects on sleep, whereas OF and 1hSD had intermediate effects. Among strains, the more anxious F344 rats exhibited the greatest sleep increases during the light period after OF, 1hSD and 4hSD. The results are discussed with respect to the relationship between behavioral and sleep responses to stressors, and to potential

mechanisms underlying the strain differences. (c) 2004 Elsevier B.V. All rights reserved.

CONCEPT CODE: Behavioral biology - General and comparative behavior 07002
Behavioral biology - Animal behavior 07003
Nervous system - Physiology and biochemistry 20504

INDEX TERMS: Major Concepts
Behavior; Nervous System (Neural Coordination)

INDEX TERMS: Diseases
anxiety: behavioral and mental disorders
Anxiety (MeSH)

INDEX TERMS: Miscellaneous Descriptors
genetic diversity; locomotion; sleep loss; acute mild stressors; rapid eye movement sleep; physiological diversity; cage change

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Wistar rat (common)
Sprague-Dawley rat (common)
Lewis rat (common)
Fischer rat (common)
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

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ACCESSION NUMBER: 2005:505791 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV200510301456
TITLE: **Ocular** surface expression of glycoprotem-340 and **surfactant protein-D.**
AUTHOR(S): Jumblatt, M. M. [Reprint Author]; Emberts, C. G.; Steele, P. S.; Jumblatt, J. E.
CORPORATE SOURCE: Univ Louisville, Louisville, KY 40292 USA
SOURCE: IOVS, (APR 2004) Vol. 45, No. Suppl. 1, pp. U565.
Meeting Info.: Annual Meeting of the Association-for-Research-in-Vision-and-Ophthalmology. Ft Lauderdale, FL, USA. April 24 -29, 2004. Assoc Res Vis & Ophthalmol.
CODEN: IOVSDA. ISSN: 0146-0404.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Nov 2005
Last Updated on STN: 23 Nov 2005

ABSTRACT:Purpose: Glycoprotein 340 (GP-340) is a member of the scavenger receptor cysteine rich group B family, acts a salivary agglutinin, and, as determined by MALDI-TOF, is present in **tears. Surfactant** ***protein*** **D (SP-D)** is a component of thelung surfactant complex and a member of the **collectin** family of proteins. **SP-D** is widely expressed in mucosal epithelia and has been detected in human and mouse lacrimal glands. **SP-** ***D*** is a mannose binding lectin and is itself bound byGP-340. The current study examines the expression of these protective proteins in ***ocular*** surface tissues including cornea, conjunctiva and lacrimal gland. In addition we examined normal human **tear** fluid for mature gp-340 and **SP-D** protein. Methods: RT-PCR was used to determine if human cornea, conjunctiva and lacrimal gland produce mRNAs

specific for gp-340 and **SP-D** as well as other known surfactant proteins (SP-A, SP-B, SP-C). Western blot analysis was used to detect the corresponding protein in human tissue extracts and **tears**. To determine the relative abundance of gp-340, Western blots of serial dilutions of tearsand saliva were evaluated. Results: Transcripts of SP-A, SP-B and SP-C were not detected in any human **ocular** tissues. ***SP*** -D and gp-340 transcripts were present in RNA extracted from cornea, conjunctiva and lacrimal gland. Western blotanalysis shows that gp-340 and **SP-D** are present in human **tear** film and that gp-340 is considerably more abundant in **tears** than in saliva. Conclusions: Thisis the first demonstration that both gp-340 and ***SP*** -D are produced by a variety of **ocular** surface mucosal tissues and that these proteins are components of the normal ***tear*** film. **SP-D** is a multifunctional, lipophilic, calcium dependentcarbohydrate binding protein, originally isolated from lung. ***SP*** -D has been shown to interact with secreted lipids, to opsonize bacterial pathogens and to modify inflammatory responses. Gp-340 binds **SP-D** and is taken up by dendritic cells and macrophages. These proteins are likely to protect the corneal and conjunctival mucosa and to link the innate and adaptive immune systems of the **ocular** surface mucosae.

CONCEPT CODE: General biology - Symposia, transactions and proceedings 00520
Genetics - General 03502
Genetics - Human 03508
Sense organs - Physiology and biochemistry 20004

INDEX TERMS: Major Concepts
Molecular Genetics (Biochemistry and Molecular Biophysics); Sense Organs (Sensory Reception)

INDEX TERMS: Parts, Structures, & Systems of Organisms
cornea: sensory system; lacrimal gland: sensory system; conjunctiva: sensory system; **tear** fluid: sensory system

INDEX TERMS: Chemicals & Biochemicals
surfactant protein-D; glycoprotein-340: expression; glycoprotein-340 mRNA [glycoprotein-340 messenger RNA]: expression; **surfactant protein-D** mRNA [**surfactant protein-D** messenger RNA]: expression; surfactant protein-A mRNA [surfactant protein-A messenger RNA]: expression; surfactant protein-B mRNA [surfactant protein-B messenger RNA]: expression; surfactant protein-C mRNA [surfactant protein-C messenger RNA]: expression

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human (common)
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

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ACCESSION NUMBER: 2005:457944 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV200510252412
TITLE: **Surfactant Protein D** is present in **tear** fluid and corneal epithelium and

inhibits corneal epithelial cell invasion by Pseudomonas aeruginosa.

AUTHOR(S): Ni, M. [Reprint Author]; Evans, D. J.; Sack, R. A.; Anders, M.; Hawgood, S.; Fleiszig, S. M. J.

CORPORATE SOURCE: Univ Calif Berkeley, Sch Optometry, Berkeley, CA 94720 USA

SOURCE: IOVS, (APR 2004) Vol. 45, No. Suppl. 2, pp. U486.
Meeting Info.: Annual Meeting of the Association-for-Research-in-Vision-and-Ophthalmology. Ft Lauderdale, FL, USA. April 24 -29, 2004. Assoc Res Vis & Ophthalmol.
CODEN: IOVSDA. ISSN: 0146-0404.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 9 Nov 2005
Last Updated on STN: 9 Nov 2005

ABSTRACT:Purpose: We have previously shown that human tear fluid protected corneal epithelial cells against P. aeruginosa (PA) in vitro and in vivo, and that protection did not depend upon tear bacteriostatic activity. We sought to identify the responsible tear component(s). The hypothesis tested was that collectins (collagenous, calcium-dependent lectins) were involved, based upon recent findings of their activities in human airway secretions.Methods: Reflex tear fluid was collected from healthy human subjects and examined for collectin content by ELISA and Western blot using antibody against surfactant ***protein*** D (SP-D), SP-A or mannose binding lectin (MBL). To test the role of detected SP-D in protection against PA invasion of corneal epithelial cells it was subtracted from human tear fluid using two separate methods; mannan-conjugated sepharose and anti-SP-D antibody. SP-D subtracted tear fluid was compared to whole team for protective effects against PA invasion using gentamicin survival assays. In other experiments, recombinant human and mouse SP-D were compared to buffer-only controls for their effect. on PA invasion. SP-***D*** distribution in the corneal epithelium of C57BL/6 mice was examined by immunofluorescence with gene-targeted C57BL/6 "knockout" mice (deficient in ***SP*** -D gene expression) used as negative controls.Results: ***SP*** -D, but not SNA or MBL, was detected by ELISA of human reflex tear fluid. Western blot analysis of whole team and of HPLC fractions confirmed thepresence of SP-D, most of which eluted in the same fraction as IgA. SP-D tearconcentrations ranged train 5 to 70 mu g/ml. Removal of SP-***D*** from human tear fluid reduced its protective activity against PA invasion ($p = 0.03$, mannan binding method; $p = 0.04$, antibody method). Both human ($p = 0.03$) and mons, ($p = 0.01$) recombinant SP-***D*** reduced PA invasion, without detectable bacteriostaticactivity. Immunoflumesence revealed SP-D antibody labeling throughout the corneal epithelium of normal, but not SP-D knockout, to ice.Conclusions: We have presented data that a mutant strain of PA defective in the early stages of biofilm formation Inks virulence in a murine model of corneal infection. This suggests that in vitro models of biofilm formation may be useful in identifying novel genes involved in the ocular virulence of PA and supports the hypothesis that there is a relationship between biofilm formation and corneal infection.

CONCEPT CODE: General biology - Symposia, transactions and proceedings 00520
Genetics - General 03502
Genetics - Animal 03506
Genetics - Human 03508
Biochemistry studies - Proteins, peptides and amino acids 10064

Sense organs - Physiology and biochemistry 20004
Physiology and biochemistry of bacteria 31000
Genetics of bacteria and viruses 31500
Immunology - General and methods 34502

INDEX TERMS: Major Concepts
Infection; Molecular Genetics (Biochemistry and Molecular Biophysics); Sense Organs (Sensory Reception)

INDEX TERMS: Parts, Structures, & Systems of Organisms
corneal epithelium: sensory system; **tear** fluid: sensory system

INDEX TERMS: Chemicals & Biochemicals
IgA [immunoglobulin A]; mannose binding lectin; **surfactant protein D**; surfactant protein-A

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human (common)
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
C57BL/6 mouse (common)
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

ORGANISM: Classifier
Pseudomonadaceae 06508
Super Taxa
Gram-Negative Aerobic Rods and Cocci; Eubacteria; Bacteria; Microorganisms
Organism Name
Pseudomonas aeruginosa (species): pathogen
Taxa Notes
Bacteria, Eubacteria, Microorganisms

GENE NAME: mouse **SP-D** gene gene [mouse protein-D gene gene] (Muridae)

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ACCESSION NUMBER: 1998:31323 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV199800031323
TITLE: Rat strain differences suggest a role for corticotropin-releasing hormone in modulating sleep.
AUTHOR(S): Opp, Mark R. [Reprint author]
CORPORATE SOURCE: Dep. Psychiatry Behavioral Sci., Univ. Texas Med. Branch, Galveston, TX 77555, USA
SOURCE: Physiology and Behavior, (Dec. 31, 1997) Vol. 63, No. 1, pp. 67-74. print.
CODEN: PHBHA4. ISSN: 0031-9384.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Jan 1998
Last Updated on STN: 14 Jan 1998

ABSTRACT:Corticotropin-releasing hormone (CRH) mediates many of the hormonal, behavioral, and autonomic responses to a variety of stressors. There is also evidence suggesting that CRH may be involved in the modulation of physiologic waking. Lewis (LEW) rats possess a hypothalamic gene defect that results in reduced synthesis and secretion of CRH relative to genetically related Fischer 344 (F344) and Sprague-Dawley (Sp-D) rat strains. We therefore hypothesized that LEW rats would spend less time awake, and more time asleep, than either F344 or Sp-D rats. Adult mate LEW, F344, and Sp-D rats were surgically provided with electroencephalograph (EEG) recording electrodes, and a thermistor to measure cortical brain temperature (T(cort)). Additional rats were also provided with a chronic guide cannula directed into a lateral cerebral ventricle. Spontaneous steep-wake behavior was determined from 48-h recordings of the EEG, T(cort), and body movements from freely behaving, undisturbed rats. Analyses of 48-h recordings from undisturbed animals indicate that LEW rats spend less time awake and more time in slow-wave sleep, relative to the other strains tested. Rapid eye movement sleep did not differ consistently between rat strains. LEW and Sp-D rats exhibit the same degree of waking in response to intracerebroventricular administration of CRH, indicating central mechanisms mediating behavioral responses to exogenously administered CRH are intact in LEW rats. These data provide support for the hypothesis that CRH may be a modulator of waking and sleep.

CONCEPT CODE: Endocrine - Pituitary 17014
Genetics - Animal 03506
Behavioral biology - Animal behavior 07003
Physiology - Stress 12008
Nervous system - Physiology and biochemistry 20504
Temperature - Thermoregulation 23012

INDEX TERMS: Major Concepts
Behavior; Endocrine System (Chemical Coordination and Homeostasis)

INDEX TERMS: Chemicals & Biochemicals
corticotropin-releasing hormone [CRH]

INDEX TERMS: Methods & Equipment
EEG [electroencephalography]: analytical method

INDEX TERMS: Miscellaneous Descriptors
cortical brain temperature; sleep modulation; strain difference; stress; waking

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rat: adult, male, strain-Fischer 344, strain-Lewis, strain-Sprague-Dawley
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

REGISTRY NUMBER: 9015-71-8 (corticotropin-releasing hormone)
9015-71-8 (CRH)

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ACCESSION NUMBER: 1997:393051 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV199799692254
TITLE: Revision of the genus Dactylozodes chevrolat 1837 (Coleoptera, Buprestidae).
AUTHOR(S): Moore, Tomas
CORPORATE SOURCE: Pirineos de Argon II, Casa 61, Curico, Chile
SOURCE: Gayana Zoologia, (1997) Vol. 61, No. 1, pp. 57-86.

CODEN: GBCZAO. ISSN: 0016-531X.
DOCUMENT TYPE: Article
(Taxonomic Key)
LANGUAGE: Spanish
ENTRY DATE: Entered STN: 10 Sep 1997
Last Updated on STN: 10 Sep 1997
ABSTRACT:The author revises the genus Dactylozodes Chevrolat, describing 12 new species: D. (s.s.) borealis n. sp., luteomarginatus n. sp., bachmanni n. ***sp.*** , D. (Parazodes) cobosi n. sp., rugicollis n. sp., acutipennis n. sp., platensis n. sp., catamarcalis n. sp., politus n. sp., bonaerensis n. sp., atrocyaneo n. sp. The twelveth, is defined as type species of a new subgenus: Arqueozodes n. subgen., with the name D. (A.) sulcatus n. sp. Also, three new subspecies: D. (s.s.) rouleti conjundatrixis n. ssp., D. (s.s.) rouleti roitmani n. ssp. and D. (P.) okea ornata n. ssp.are described. The genus Agrilozodes Thery is revalidated for the species **ocularis** Kerr., pygmaea Kerr. and praeclara Perroud. A key for all the species is presented.
CONCEPT CODE: General biology - Taxonomy, nomenclature and terminology 00504
Anatomy and Histology - Gross anatomy 11102
Animal distribution - 62800
Invertebrata: general and systematic - Insecta: Coleoptera 63573
Invertebrata: comparative, experimental morphology, physiology and pathology - Insecta: morphology, comparative 64074
INDEX TERMS: Major Concepts
Biogeography (Population Studies); General Life Studies; Morphology; Systematics and Taxonomy
INDEX TERMS: Miscellaneous Descriptors
SYSTEMATICS
ORGANISM: Classifier
Coleoptera 75304
Super Taxa
Insecta; Arthropoda; Invertebrata; Animalia
Organism Name
Buprestidae
Dactylozodes
Dactylozodes acutipennis: new species
Dactylozodes atrocyaneo: new species
Dactylozodes bachmanni: new species
Dactylozodes bonaerensis: new species
Dactylozodes borealis: new species
Dactylozodes catamarcalis: new species
Dactylozodes cobosi: new species
Dactylozodes luteomarginatus: new species
Dactylozodes okea ornata: new subspecies
Dactylozodes platensis: new species
Dactylozodes politus: new species
Dactylozodes rouleti conjundatrixis: new subspecies
Dactylozodes rouleti roitmani: new subspecies
Dactylozodes rugicollis: new species
Dactylozodes sulcatus: new species
Taxa Notes
Animals, Arthropods, Insects, Invertebrates
ORGANISM: Classifier
Organisms 00500
Super Taxa
Organisms
Organism Name

Arqueozodes: new subgenus
Taxa Notes
Organisms

L148 ANSWER 36 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:43563 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV200600052764
TITLE: Pseudomonas aeruginosa exposure regulates **surfactant protein D** production by human corneal epithelial cells.
AUTHOR(S): Ni, M. [Reprint Author]; Evans, D. J.; Fleiszig, S. M. J.
SOURCE: IOVS, (2005) Vol. 46, No. Suppl. S, pp. 896.
Meeting Info.: Annual Meeting of the Association-for-Research-in-Vision-and-Ophthalmology. Ft Lauderdale, FL, USA. May 01 -05, 2005. Assoc Res Vis & Ophthalmol.
CODEN: IOVSDA. ISSN: 0146-0404.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Jan 2006
Last Updated on STN: 4 Jan 2006

ABSTRACT:Purpose: We previously showed that **SP-D** was present in human **tear** fluid and that it protected corneal epithelial cells against Pseudomonas aeruginosa invasion in vitro. In this study, we explored the expression of **SP-D** in corneal epithelium and then examined the effect of P. aeruginosa exposure. Methods: Primary cultures of mouse corneal epithelial cells were prepared from female 8-12 week old wild type C57BL/6 mice and gene-targeted **SP-D** deficient mice. SDS-PAGE and Western blot were performed to detect the **SP-D** level in cultured corneal epithelial cell lysates or cell growth media. To determine whether P. aeruginosa exposure can regulate **SP-D** expression, SV 40-immortalized human corneal epithelial cells were stimulated with 2 x 107 or 2 x 1010 heat killed bacteria (invasive strain PAK) or were sham inoculated. After stimulation, cells were lysed and **SP-**
D quantified using Western Blot. The effect of bacterial lipopolysaccharide (LPS) on **SP-D** expression was explored using two different mutants withdefects in LPS core and O antigen. A fliC mutant was used to examine the roleof bacterial flagellin. Results: **SP-D** was detected in primary cultured mouse corneal epithelial cells derived from C57BL/6 mice, but not in lysates of corneal epithelial cells derived from **SP-D** deficient mice. **SP-D** was also detected extracellularly in cultured corneal epithelial cell growth media. Cells treated with heat killed wild type P. aeruginosa showed a strong dose-dependent upregulation of **SP-D** production in both cell lysates and cellular secretions. LPS and flagellin mutants, however, were each defective in their ability to upregulate **SP-D** in cell lysates and cell secretions. Conclusions: Corneal epithelialcells were found to make and secrete **SP-D** and as such could contribute to
tear fluid **SP-D** level. **SP-D** expression in human corneal epithelial cells was strongly upregulated when cells were exposed to P. aeruginosa, which involved bacterial LPS and flagellin. These results suggest that **SP-D** is an inducible factor involved in innate immunity against P. aeruginosa invasion, and that induction could involve TLR signaling.

CONCEPT CODE: General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Cytology - Human 02508
Biochemistry studies - General 10060

Sense organs - Physiology and biochemistry 20004
Physiology and biochemistry of bacteria 31000

INDEX TERMS: Major Concepts
Biochemistry and Molecular Biophysics; Sense Organs (Sensory Reception)

INDEX TERMS: Parts, Structures, & Systems of Organisms
corneal epithelial cell: sensory system

INDEX TERMS: Chemicals & Biochemicals
flagellin; lipopolysaccharide O antigen; TLR: signaling; **surfactant protein D**:
expression, production; lipopolysaccharide core antigen

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human (common)
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
C57BL/6 mouse (common): female
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

ORGANISM: Classifier
Pseudomonadaceae 06508
Super Taxa
Gram-Negative Aerobic Rods and Cocci; Eubacteria; Bacteria; Microorganisms
Organism Name
Pseudomonas aeruginosa (species)
Taxa Notes
Bacteria, Eubacteria, Microorganisms

L148 ANSWER 37 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:291248 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV200400290730
TITLE: Heterogeneous allele expression of pulmonary surfactant protein (SP)-A and Osteopontin (OPN) in rat.
AUTHOR(S): Lin, Zhenwu [Reprint Author]; Wang, Yunhua; Zhu, Kangmin; Floros, Joanna
CORPORATE SOURCE: Cellular and Molecular Physiology, The Penn State Univ College of Medicine, H166, 500 Univ Drive, Hershey, PA, 17033, USA
zlin@psu.edu
SOURCE: FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 174.15. http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English

ENTRY DATE: Entered STN: 23 Jun 2004
Last Updated on STN: 23 Jun 2004

ABSTRACT:SP-A and OPN play roles in innate host defense and regulation of inflammatory processes. These genes are expressed in rat in a tissue-specific manner. SP-A is expressed predominantly in lung, and also in small and large intestine, **eye**, ear, stomach, vagina, and penis, not in brain, tongue, heart, liver, kidney, and spleen, whereas OPN is expressed in all of the 14 tissues and predominantly in kidney. Allele expression of SP-A and OPN exhibits a heterogeneous pattern. SP-A (n=66)exhibits exclusively balanced biallelic (BB)expression in the lung, and with 59% BB and 41% imbalanced biallelic (IB) in the large intestines. Allele expression of OPN is more heterogeneous. In the large intestine, OPN (n=59) exhibits a predominant IB expression (64%), followed by BB (22%) and monoallelic (MO) expression (14%). The allele expression of OPN was compared among five tissues from a single rat pedigree. The results showed that allele expression was tissue specific. A pedigree study of OPN allele expression suggested that inheritable factor(s) are involved in the regulation of allele expression. Analysis of co-expression of alleles of these genes from double heterozygous rats for any two of SP-A, ***SP*** **-D**, and OPN showed independent allele regulation. We conclude that the expression of the alleles of these 3 genes is not coordinately regulated. Supported by NIH R37HL34788, AHA 198312P and PSU DFG.

CONCEPT CODE:
General biology - Symposia, transactions and proceedings 00520
Genetics - General 03502
Genetics - Animal 03506
Biochemistry studies - Proteins, peptides and amino acids 10064
Digestive system - Physiology and biochemistry 14004
Cardiovascular system - Physiology and biochemistry 14504
Blood - Blood and lymph studies 15002
Blood - Blood cell studies 15004
Urinary system - Physiology and biochemistry 15504
Respiratory system - Physiology and biochemistry 16004
Reproductive system - Physiology and biochemistry 16504
Dental biology - Physiology and biochemistry 19004
Sense organs - Physiology and biochemistry 20004
Nervous system - Physiology and biochemistry 20504
Immunology - General and methods 34502

INDEX TERMS:
Major Concepts
Blood and Lymphatics (Transport and Circulation); Cardiovascular System (Transport and Circulation); Dental and Oral System (Ingestion and Assimilation); Digestive System (Ingestion and Assimilation); Immune System (Chemical Coordination and Homeostasis); Molecular Genetics (Biochemistry and Molecular Biophysics); Nervous System (Neural Coordination); Reproductive System (Reproduction); Sense Organs (Sensory Reception); Urinary System (Chemical Coordination and Homeostasis)

INDEX TERMS:
Parts, Structures, & Systems of Organisms
allele; brain: nervous system; ear: sensory system; **eye**: sensory system; heart: circulatory system; kidney: excretory system; large intestine: digestive system; liver: digestive system; lung: respiratory system; penis: reproductive system; spleen: blood and lymphatics, immune system; tongue: dental and oral system; vagina: reproductive system

INDEX TERMS:
Chemicals & Biochemicals
osteopontin: expression, heterologous allelic expression, pulmonary; surfactant protein-A: expression,

heterologous allelic expression, pulmonary

INDEX TERMS: Miscellaneous Descriptors
inflammatory process regulation; innate host defense

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rat (common)
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

GENE NAME: mouse **SP-D** gene [mouse **surfactant protein-D** gene] (Muridae); rat OPN gene [rat osteopontin gene] (Muridae); rat SP-A gene [rat surfactant protein-A gene] (Muridae)

L148 ANSWER 38 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:607222 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV200200607222
TITLE: Heterogeneous allele expression of pulmonary surfactant protein (**SP**)-**D** gene in rat large intestine and other tissues.
AUTHOR(S): Lin, Z. [Reprint author]; Floros, J. [Reprint author]
CORPORATE SOURCE: Dept Cell Mol Physiol, Penn State Univ college Med, Hershey, PA, USA
SOURCE: American Journal of Human Genetics, (October, 2002) Vol. 71, No. 4 Supplement, pp. 336. print.
Meeting Info.: 52nd Annual Meeting of the American Society of Human Genetics. Baltimore, MD, USA. October 15-19, 2002. American Society of Human Genetics.
CODEN: AJHGAG. ISSN: 0002-9297.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Nov 2002
Last Updated on STN: 27 Nov 2002
CONCEPT CODE: General biology - Symposia, transactions and proceedings 00520
Genetics - General 03502
Genetics - Animal 03506
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Physiology - General 12002
Digestive system - Physiology and biochemistry 14004
Cardiovascular system - Physiology and biochemistry 14504
Blood - Blood and lymph studies 15002
Blood - Blood cell studies 15004
Urinary system - Physiology and biochemistry 15504
Respiratory system - Physiology and biochemistry 16004
Reproductive system - Physiology and biochemistry 16504
Dental biology - Physiology and biochemistry 19004
Sense organs - Physiology and biochemistry 20004
Nervous system - Physiology and biochemistry 20504
Immunology - General and methods 34502
INDEX TERMS: Major Concepts
Molecular Genetics (Biochemistry and Molecular Biophysics); Physiology
INDEX TERMS: Parts, Structures, & Systems of Organisms

brain: nervous system; ear: sensory system; eye : sensory system; heart: circulatory system; kidney: excretory system; large intestine: digestive system; liver: digestive system; lung: respiratory system; penis: reproductive system; small intestine: digestive system; spleen: blood and lymphatics, immune system; tongue: dental and oral system; vagina: reproductive system

INDEX TERMS: Methods & Equipment
Northern blot: analytical method, blotting/hybridization/molecular probe techniques, gene mapping, genetic method, labeling, recombinant DNA technology; reverse transcriptase-polymerase chain reaction: analytical method, genetic method, polymerase chain reaction

INDEX TERMS: Miscellaneous Descriptors
Meeting Abstract

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rat
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

GENE NAME: rat **SP-D** gene [rat **surfactant protein-D** gene] (Muridae): heterogeneous allele expression, tissue specific expression

L148 ANSWER 39 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:408134 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV199800408134
TITLE: The discovery of Early Ordovician microfossils from the Sujiane Group of Tongbo-Dabie Orogenic Belt, and its significances.
AUTHOR(S): Zhang, Ren-Jie; Chen, Xiao-Hong [Reprint author]
CORPORATE SOURCE: Yichang Inst. Geol. Mineral Resources, Yichang 443003, China
SOURCE: Acta Micropalaeontologica Sinica, (June, 1998) Vol. 15, No. 2, pp. 125-133. print.
ISSN: 1000-0674.
DOCUMENT TYPE: Article
LANGUAGE: Chinese
ENTRY DATE: Entered STN: 21 Sep 1998
Last Updated on STN: 21 Sep 1998

ABSTRACT:The Sujiahe Group, including Huwan and Dingyuan Formations named by Beijing College of Geology in 1960, has a wide E-W oriented distribution in Tongbo-Dabie Orogenic Belt (Henan part). It also is the one of the major members of that orogenic belt, which together with the Qinling Orogenic Belt separates the Huabei (North China) and Yangtze plates. The Dingyuan Formation (upper part of Sujiahe Group) is a suit of lower grade greenschist and volcanic rocks, about 920-1470 m thick. The Huwan Formation (lower part of Sujiahe Group) consists of mid-high grade metamorphozed rocks with a thickness of 1540 -3000 m, including two members. The upper member consists mainly of gneiss, intercalated with few marble beds, plagi-amphibole schist, quartzite and muscovite-quartz schist. The lower member is composed of clastic and muddy-calcareous deposits containing some marbles. The contact between the Dingyuan and Huwan Formations used to be thought as a parallel unconformity.

the Sujiahe Group angular-unconformably overlies rocks of the Qijiaoshan Formation, Hongan Group. The contact between the Sujiahe Group and overlying Xinyang Group is a fault. However, the Huwan Formation, Sujiahe Group at Xishuanhe area, Xinyang City, consists mainly of muscovite-plagiogneiss, calcite-marble, muscovite-marble, albite-leucoleptynite, and muscovite-schist, 1862 m thick. The lower part of that formation intercalates with lens of half graphite quartz schist, from where the microfossils including chitinozoans and acritarchs have been found (Fig. 1). The chitinozoans include Conochitina sp., Desmochitina sp., D. brechyta, Lagenochitina cf. obeligis, L. cf. esthonica, Rhabdochitina sp., and Calpichitina? sp., while the acritarchs comprise Leiosphaeridia spp. , Lophoshaeridium sp., Micrhystridium sp.? Acanthodiacrodium sp., Balthisphaeridium spp. The discovery of microfossils in the Huwan Formation may be one of the great advances in the research on the stratigrap by and paleontology in Tongbo-Dabie Orogenic Belt. It might exert a tremendous impact on the future tectonic research, regional geological survey and ore-prospecting in that region. The major significant points are 1. Determination of the geological age of Huwan Formation, Sujiahe Group. Because no fossils were previously found, according to the geological setting and the degree of metamorphism, the Sujiahe Group used to be assigned to Proterozoic. On the basis of the occurrence of such chitinozoans as Lagenochitina cf. esthonica, L. cf. obeligis and Desmochitina brechyta the fossil-bearing rocks, the Huwan Formation is roughly assignable to the Arenigian Stage. The acritarchs also suggest a Cambrian-Ordovician age. The Lower Ordovician age with fossil evidence is recognized for the first time in Tongbo-Dabie Oro enic Belt, although earlier reports of Ordovician fossils were from thin-sections of marble of the Huwan Formation, Sujiahe Group, in Xiongdian Village, Lushan County, Henan Province, and Xuanhuadian Town, Dawu County, Hubei Province (Ye et al., 1991, 1993). Nevertheless, no fossil lists which may indicate the Ordovician age have been released, except the very rough fossil classifications such as brachiopods, stems of crinoids, foraminiferas which range from Cambrian to recent. Obviously, such evidence for judging Ordovician strata of Huwan Formation was too tenuous. 2. The discovery of Early Ordovician offers new information for the establishment of regional stratigraphic sequence in the Tongbo - Dabie Orogenic Belt. It also offers some information for reinterpreting the tectonic evolution of that orogenic belt, and the connection between the Qinling and Tongbo-Dabie Orogenic Belts. 3. The paleobiogeographic significance: the chitinozoans of Huwan Formation such as Calpichitina sp., Desmochitina brechyta, Lagenochitina obeligis, and L. esthonica are the common members of contemporaneous chitinozoans of South China. Hence, it is reasonable to place them in the same paleobiogeographic region.

CONCEPT CODE: Paleozoology - 63000
Paleobotany - 50000
Botany: general and systematic - Algae 50504
Invertebrata: general and systematic - Mollusca 63526
Geological periods - Ordovician 64708
INDEX TERMS: Major Concepts
Paleobiology
INDEX TERMS: Time
Ordovician
INDEX TERMS: Miscellaneous Descriptors
biostratigraphy; paleogeography; Dabie Orogenic Belt; Fossil
GEOGRAPHICAL TERMS: Sujiahe Group (China, Asia, Palearctic region)
ORGANISM: Classifier
Loricata 61300
Super Taxa
Mollusca; Invertebrata; Animalia
Organism Name
Calpichitina-sp. [chitinozoan]: Fossil

Conochitina-sp. [chitinozoan]: Fossil
Desmochitina-brechyta [chitinozoan]: Fossil
Desmochitina-sp. [chitinozoan]: Fossil
Lagenochitina-esthonica [chitinozoan]: Fossil
Lagenochitina-obeligis [chitinozoan]: Fossil
Rhabdochitina-sp. [chitinozoan]: Fossil
Taxa Notes
Animals, Invertebrates, Mollusks

ORGANISM:
Classifier
Pyrrophyta 14500
Super Taxa
Algae; Plantae
Organism Name
Acanthodiacrodium-sp. [acritarch]: Fossil
Balthisphaeridium-sp. [acritarch]: Fossil
Leiosphaeridia-spp. [acritarch]: Fossil
Lophoshaeridium-sp. [acritarch]: Fossil
Micrhystridium-sp. [acritarch]: Fossil
Taxa Notes
Algae, Microorganisms, Nonvascular Plants, Plants

L148 ANSWER 40 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1984:181254 BIOSIS Full-text<<LOGINID::20061004>>
DOCUMENT NUMBER: PREV198477014238; BA77:14238
TITLE: IN THE **EYE** 2-D PROLINE-7 9-D-TRYPTOPHAN SUBSTANCE P IS A SUBSTANCE P AGONIST WHICH MODIFIES THE RESPONSES TO SUBSTANCE P PROSTAGLANDIN E-1 AND ANTIDROMIC TRIGEMINAL NERVE STIMULATION.
AUTHOR(S): MANDAHL A [Reprint author]; BILL A
CORPORATE SOURCE: DEP OPHTHALMOL, UNIV HOSP, UPPSALA, SWEDEN
SOURCE: Acta Physiologica Scandinavica, (1983) Vol. 117, No. 1, pp. 139-144.
CODEN: APSCAX. ISSN: 0001-6772.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

ABSTRACT:A substance P analog, (D-Pro2, D-Trp7,9)-**SP** [D-2-proline-D-7,9-tryptophan substance P] was described to have SP antagonistic and SP agonistic effects in different tissues. The effects of (D-Pro2, D-Trp7,9)-SP was investigated on the sphincter pupillae muscle, the blood aqueous barrier (BAB) and the **intraocular** pressure (IOP) in the albino rabbit **eye**. The modifying effects of (D-Pro2, D-Trp7,9)-SP on miosis, BAB damage and IOP rise caused by SP, prostaglandin E1 (PGE1), capsaicin and on the miosis caused by electrical intracranial antidromic trigeminal nerve stimulation (NV stim). Endogenous PG synthesis was inhibited by systemic indomethacin i.v., cholinergic influence on the pupil size was inhibited with biperiden, i.v., adrenergic nerve influence by cervical sympathectomy just prior to the experiments. Tubocurarine chloride was used to cause relaxation of striated muscles in the experiments with NV stim. D-Pro2, D-Trp7,9-SP (100 µg) to cause miosis, breakdown of the BAB with heavy leakage of Evans blue into the ciliary processes and aqueous humor, and a rise in IOP. At 10 µg (D-Pro2, D-Trp7,9)-SP caused slight miosis and did not inhibit the miosis caused by SP or capsaicin, but caused a significant reduction of the miotic response caused by PGE1 and NV stim. The rise in protein concentration in the aqueous humor caused by SP or PGE1 was slightly but significantly lower after pretreatment with (D-Pro2, D-Trp7,9)-SP. D-Pro2, D-Trp7,9-SP was found to act as a SP agonist on the sphincter pupillae muscle, on the BAB and IOP. D-Pro2, D-Trp7,9-SP seemed to have some SP antagonistic effects on mechanisms that require sensory nerve conduction, e.g., miosis

caused by PGE1 and NV stim. The antagonistic mechanism is not clear. The SP analog may have an unspecific membrane stabilizing effect or a toxic effect or block SP receptors on the sensory nerve fibers. Such effects of (D-Pro2, D-Trp7,9)-SP may explain also, why the rise in protein concentration in the aqueous humor caused by SP and PGE1 was lower in **eyes** pretreated with (D-Pro2, D-Trp7,9)-SP.

CONCEPT CODE: Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Lipids 10066
Enzymes - Methods 10804
Metabolism - Lipids 13006
Metabolism - Proteins, peptides and amino acids 13012
Cardiovascular system - Physiology and biochemistry 14504
Blood - Other body fluids 15010
Endocrine - General 17002
Endocrine - Neuroendocrinology 17020
Muscle - General and methods 17501
Muscle - Physiology and biochemistry 17504
Muscle - Pathology 17506
Sense organs - General and methods 20001
Sense organs - Physiology and biochemistry 20004
Sense organs - Pathology 20006
Nervous system - General and methods 20501
Nervous system - Physiology and biochemistry 20504
Nervous system - Pathology 20506
Pharmacology - Neuropharmacology 22024
Pharmacology - Sense organs, associated structures and functions 22031

INDEX TERMS: Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry and Molecular Biophysics); Metabolism; Muscular System (Movement and Support); Nervous System (Neural Coordination); Pharmacology; Sense Organs (Sensory Reception)

INDEX TERMS: Miscellaneous Descriptors
RABBIT **OPHTHALMIC**-DRUG SPHINCTER PUPILLAE MUSCLE BLOOD AQUEOUS BARRIER CILIARY PROCESS INTRA **OCULAR** PRESSURE CAPSAICIN MIOSIS PROTEIN CONCENTRATION ELECTRICAL NERVE STIMULATION INDOMETHACIN BIPERIDEN SENSORY NERVE CONDUCTION

ORGANISM: Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates

REGISTRY NUMBER: 33507-63-0 (SUBSTANCE P)
745-65-3 (PROSTAGLANDIN E1)
404-86-4 (CAPSAICIN)
53-86-1 (INDOMETHACIN)
514-65-8 (BIPERIDEN)

L148 ANSWER 41 OF 45 DISSABS COPYRIGHT (C) 2006 ProQuest Information and Learning Company; All Rights Reserved on STN

ACCESSION NUMBER: 1999:33066 DISSABS Order Number: AAR9903270

TITLE: **EYE** OF THE PUGILIST: CHARACTERIZATION OF A DOMINANT MUTATION THAT CAUSES PATTERNED AND VARIEGATED DEFECTS IN DROSOPHILA **EYE** PIGMENTATION (DROSOPHILA MELANOGASTER)

AUTHOR: RONG, YIKANG [PH.D.]; GOLIC, KENT G. [adviser]
CORPORATE SOURCE: THE UNIVERSITY OF UTAH (0240)
SOURCE: Dissertation Abstracts International, (1998) Vol. 59, No. 8B, p. 3872. Order No.: AAR9903270. 156 pages.
DOCUMENT TYPE: Dissertation
FILE SEGMENT: DAI
LANGUAGE: English
ABSTRACT:

This dissertation describes the isolation and characterization of a eye color mutation in Drosophila melanogaster. It presents a story that may be of general interest for a number of fields, namely, eye development, metabolic genes, and chromosomal position effects.

The $pugilist\sp{Dominant}$($pug***sp*** { D}$) mutation causes a dominant and patterned defect in eye pigmentation. In a genetic background that only allows pteridine pigment synthesis, pigmentation is limited to the eye periphery so that the eye has a striking ring of red pigment, with a white center. A small and variable number of cells in the middle of the eye possess normal pigmentation, showing the variegating aspect of the pug phenotype. The mutant $pug***sp*** {D}$ gene is a fusion between a stretch of highly repetitive DNA sequences (the GAGA repeats) and part of a gene whose product is the trifunctional methylenetetrahydrofolate dehydrogenase (MTHFD). The MTHFD enzyme provides essential functions in purine de novo synthesis. This may be related to the pteridine defects in $pug***sp*** {D }$, since pteridines are synthesized from purine precursors.

We have tested several hypotheses for the mechanism underlying the pug phenotype. This was partly accomplished by using the FLP-mediated DNA mobilization technique. The successful development of this technique allows targeting of a gene to numerous chromosomal locations without mutating the gene of interest. The technique also enables targeting of many different genes to a specific locus, thus eliminating the variable of position effects. Armed with this powerful technique, we showed that $pug***sp*** { D}$ is highly sensitive to position effects. The phenotype may be influenced by the level of $pug***sp*** {D}$ expression, and/or by the distance between the $pug***sp*** {D}$ locus and centric heterochromatin. It was also shown that the length of the GAGA repeats affects the phenotype. Interestingly, translation of the repeats is necessary for the phenotype. It seems that a shorter repeat would generate a weaker pug phenotype. A few models have been proposed for the pug phenotype. First, the novel $pug***sp*** {D}$ protein could disrupt purine synthesis by interfering with the functions of MTHFD. Second, this purine defect could knock out pteridine synthesis, and be responsible for the ring pattern of pigmentation in that only cells at the periphery could take up enough exogenous purines for pigment synthesis. Third, the variegating aspect of the phenotype may invole position effect variegation (PEV). We proposed that the occasional pigmented spots in the middle of the eye reflect instances of $pug***sp*** {D}$ inactivation. This gene silencing may be brought about

by the ectopic association between the heterochromatic GAGA repeats in $pug***sp*** {D }$ and centric heterochromatin. If true, this would be the smallest piece of heterochromatin yet identified that causes PEV.

CLASSIFICATION: 0369 BIOLOGY, GENETICS; 0307 BIOLOGY, MOLECULAR

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ACCESSION NUMBER: 96:52124 DISSABS Order Number: AAR9626711

TITLE: NUCLEAR ARCHITECTURE IN DROSOPHILA MELANOGASTER (FLUORESCENCE IN SITU HYBRIDIZATION, CHROMOSOME ORGANIZATION)

AUTHOR: DERNBURG, ABBY FELICIA [PH.D.]; SEDAT, JOHN W. [advisor]

CORPORATE SOURCE: UNIVERSITY OF CALIFORNIA, SAN FRANCISCO (0034)

SOURCE: Dissertation Abstracts International, (1996) Vol. 57, No. 4B, p. 2276. Order No.: AAR9626711. 316 pages.

DOCUMENT TYPE: Dissertation

FILE SEGMENT: DAI

LANGUAGE: English

ENTRY DATE: Entered STN: 19960903
Last Updated on STN: 19960903

ABSTRACT: This dissertation describes the development of novel techniques for fluorescence in situ hybridization (FISH) in whole-mount tissues and their application to the study of chromosome organization in Drosophila melanogaster. The methodology is described in detail in the first chapter. Each of the subsequent chapters presents experiments using FISH to address a particular question in chromosome biology.

One study investigates the relationship between the global arrangement of chromosomes in the interphase nucleus and the expression of an eye-color gene, brown, which displays dominant position-effect variegation(PEV). Using three-dimensional FISH and statistics, I show that the mutation $bw***sp*** {D}$, which was caused by insertion of a block of heterochromatin near the end of a chromosome, markedly alters the normal organization of the interphase nucleus. This impact of the $bw***sp*** {D}$ mutation on nuclear architecture is due to specific physical interactions between the insertion and centromeric heterochromatin in an intrachromosomal fashion.

Moreover, this work provides some of the most compelling evidence to date of dramatic differences in chromosome organization as a function of the development of an organism. A separate study explores the issue of chromosome paining during meiosis in Drosophila oocytes. Specifically, I used FISH to investigate the mechanism of achiasmate (or "distributive") segregation by asking whether chromosomes that fail to undergo exchange with a homolog are nevertheless paired with a partner chromosome. This work illustrated that pairwise associations are maintained between homologous chromosomes even without crossing over, but that pairs of heterologous chromosomes can segregate from each other without prior association. In the final chapter, I examine a perplexing example of non-Mendelian inheritance through the male germ line in Drosophila. A particular compound chromosome, C(2)EN, is transmitted only poorly to the progeny of males that carry it. In

collaboration with other investigators, I showed that this phenomenon is an example of meiotic drive, in which sperm containing the compound chromosome undergo selective dysfunction at a particular stage of the fertilization process. A reiterated theme throughout these studies is the key role of heterochromatin in the organization of Drosophila chromosomes in both somatic and meiotic nuclei.

CLASSIFICATION: 0379 BIOLOGY, CELL; 0369 BIOLOGY, GENETICS; 0307 BIOLOGY, MOLECULAR

L148 ANSWER 43 OF 45 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:633983 SCISEARCH Full-text<<LOGINID::20061004>>
THE GENUINE ARTICLE: 836JQ
TITLE: Surfactant proteins A and D enhance the phagocytosis of Chlamydia into THP-1 cells
AUTHOR: Oberley R E; Ault K A; Neff T L; Khubchandani K R; Crouch E C; Snyder J M (Reprint)
CORPORATE SOURCE: Univ Iowa, Coll Med, Dept Anat & Cell Biol, Iowa City, IA 52242 USA (Reprint); Univ Iowa, Coll Med, Dept Obstet & Gynecol, Iowa City, IA 52242 USA; Washington Univ, Sch Med, Dept Pathol, St Louis, MO 63110 USA
jeanne-snyder@uiowa.edu
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-LUNG CELLULAR AND MOLECULAR PHYSIOLOGY, (AUG 2004) Vol. 287, No. 2, pp. L296-L306. ISSN: 1040-0605.
PUBLISHER: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
DOCUMENT TYPE: Article; Journal
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REFERENCE COUNT: 48
ENTRY DATE: Entered STN: 6 Aug 2004
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ABSTRACT:
Chlamydiae are intracellular bacterial pathogens that infect mucosal surfaces, i.e., the epithelium of the lung, genital tract, and conjunctiva of the **eye**, as well as alveolar macrophages. In the present study, we show that pulmonary surfactant protein A (SP-A) and **surfactant** ***protein*** **D (SP-D)**, lung **collectins** involved in innate host defense, enhance the phagocytosis of Chlamydia pneumoniae and Chlamydia trachomatis by THP-1 cells, a human monocyte/macrophage cell line. We also show that SP-A is able to aggregate both C. trachomatis and C. pneumoniae but that **SP-D** only aggregates C. pneumoniae. In addition, we found that after phagocytosis in the presence of SP-A, the number of viable C. trachomatis pathogens in the THP-1 cells 48 h later was increased similar to3.5-fold. These findings suggest that SP-A and **SP-D** interact with chlamydial pathogens and enhance their phagocytosis into macrophages. In addition, the chlamydial pathogens internalized in the presence of **collectins** are able to grow and replicate in the THP-1 cells after phagocytosis.

CATEGORY: PHYSIOLOGY; RESPIRATORY SYSTEM
SUPPLEMENTARY TERM: SP-A; **SP-D**; Chlamydia trachomatis; Chlamydia pneumoniae
SUPPL. TERM PLUS: ALVEOLAR MACROPHAGES; PSEUDOMONAS-AERUGINOSA; DEFICIENT MICE; IN-VITRO; MYCOBACTERIUM-TUBERCULOSIS; PNEUMONIAE; INFECTION; TRACHOMATIS; AGGREGATION; CLEARANCE

REFERENCE(S):

Referenced Author	Year	VOL	ARN PG	Referenced Work

(RAU)	(RPY)	(RVL)	(RPG)	(RWK)
BEHARKA A A	2002	169	3565	J IMMUNOL
BRADFORD M M	1976	72	248	ANAL BIOCHEM
CARRATELLI C R	2002	215	69	FEMS MICROBIOL LETT
CROUCH E	2001	63	521	ANNU REV PHYSIOL
CROUCH E	1994	269	15808	J BIOL CHEM
DASILVA O	1997	16	364	PEDIATR INFECT DIS J
EJZENBERG B	1996	38	9	REV INST MED TROP SP
FERGUSON J S	2002	168	1309	J IMMUNOL
FRICK I M	2000	37	1232	MOL MICROBIOL
GAYDOS C A	1996	64	1614	INFECT IMMUN
HAAGSMAN H P	1998	1408	264	BBA-MOL BASIS DIS
HAAGSMAN H P	2001	129	91	COMP BIOCHEM PHYS A
HACKSTADT T	1999		101	CHLAMYDIA INTRACELLU
HARTSHORN K L	2002	70	6129	INFECT IMMUN
HARTSHORN K L	1998	274	L958	AM J PHYSIOL-LUNG C
HATCH T P	1999		29	CHLAMYDIA INTRACELLU
HESS S	2001	44	2392	ARTHRITIS RHEUM
HICKLING T P	1999	29	3478	EUR J IMMUNOL
JAIN S	1999	8	130	J MATERN FETAL MED
JOHANSSON J	1997	244	675	EUR J BIOCHEM
KHUBCHANDANI K R	2001	25	699	AM J RESP CELL MOL
KNUTTON S	1999	33	499	MOL MICROBIOL
KOSMA P	1999	1455	387	BBA-MOL BASIS DIS
LEVINE A M	2000	165	3934	J IMMUNOL
LEVINE A M	1999	20	279	AM J RESP CELL MOL
LEVINE A M	1998	19	700	AM J RESP CELL MOL
LEVINE A M	1997	158	4336	J IMMUNOL
LEVINE A M	2001	167	5868	J IMMUNOL
LEVINE A M	1999	103	1015	J CLIN INVEST
MADAN T	2001	69	2728	INFECT IMMUN
MCCORMACK F X	1998	1408	109	BBA-MOL BASIS DIS
MOAZED T C	1998	177	1322	J INFECT DIS
OFEK I	2001	69	24	INFECT IMMUN
PAUL T R	1997	39	623	J ANTIMICROB CHEMOTH
PIKAAR J C	1995	172	481	J INFECT DIS
REDECKE V	1998	19	721	AM J RESP CELL MOL
REID K B M	1998	1408	290	BBA-MOL BASIS DIS
RESTREPO C I	1999	21	576	AM J RESP CELL MOL
SCHACHTER J	1999		139	CHLAMYDIA INTRACELLU
STOKES R W	1999	197	1	CELL IMMUNOL
SZABO E	1998	12	403	INT J ONCOL
THIELE L	2001	76	59	J CONTROL RELEASE
TINO M J	1996	270	677	AM J PHYSIOL
VANDEWETERING J K	2001	184	1143	J INFECT DIS
VANROZENDAAL B A W M	2000	182	917	J INFECT DIS
WEIKERT L F	2000	279	L216	AM J PHYSIOL-LUNG C
WRIGHT J R	1997	77	931	PHYSIOL REV
YONG S J	2003	71	1662	INFECT IMMUN

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ACCESSION NUMBER: 2004:581460 SCISEARCH Full-text<<LOGINID::20061004>>

THE GENUINE ARTICLE: 829YT

TITLE: Microarray analysis of the rat lacrimal gland following the loss of parasympathetic control of secretion

AUTHOR: Nguyen D H; Toshida H; Schurr J; Beuerman R W (Reprint)

CORPORATE SOURCE: Louisiana State Univ, Ctr Eye, Lions Eye Res Labs, Lab Mol Biol Ocular Surface, 2020 Gravier St, Suite B, New

Orleans, LA 70112 USA (Reprint); Louisiana State Univ, Ctr Eye, Lions Eye Res Labs, Lab Mol Biol Ocular Surface, New Orleans, LA 70112 USA; Louisiana State Univ, Hlth Sci Ctr, Dept Genet, GeneChip Bioinformat Core, New Orleans, LA 70112 USA; Singapore Eye Res Inst, Singapore 168751, Singapore
rbeuer@lsuhsc.edu
COUNTRY OF AUTHOR: USA; Singapore
SOURCE: PHYSIOLOGICAL GENOMICS, (17 JUN 2004) Vol. 18, No. 1, pp. 108-118.
ISSN: 1094-8341.
PUBLISHER: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 76
ENTRY DATE: Entered STN: 16 Jul 2004
Last Updated on STN: 16 Jul 2004

ABSTRACT:
Previous studies showed that loss of muscarinic parasympathetic input to the lacrimal gland (LG) leads to a dramatic reduction in **tear** secretion and profound changes to LG structure. In this study, we used DNA microarrays to examine the regulation of the gene expression of the genes for secretory function and organization of the LG. Long-Evans rats anesthetized with a mixture of ketamine/xylazine (80:10 mg/kg) underwent unilateral sectioning of the greater superficial petrosal nerve, the input to the pterygopalatine ganglion. After 7 days, **tear** secretion was measured, the animals were killed, and structural changes in the LG were examined by light microscopy. Total RNA from control and experimental LGs (n = 5) was used for DNA microarray analysis employing the U34A GeneChip. Three statistical algorithms (detection, change call, and signal log ratio) were used to determine differential gene expression using the Microarray Suite (5.0) and Data Mining Tools (3.0). **Tear** secretion was significantly reduced and corneal ulcers developed in all experimental **eyes.** Light microscopy showed breakdown of the acinar structure of the LG. DNA microarray analysis showed downregulation of genes associated with the endoplasmic reticulum and Golgi, including genes involved in protein folding and processing. Conversely, transcripts for cytoskeleton and extracellular matrix components, inflammation, and apoptosis were upregulated. The number of significantly upregulated genes (116) was substantially greater than the number of downregulated genes (49). Removal of the main secretory input to the rat LG resulted in clinical symptoms associated with severe dry **eye** . Components of the secretory pathway were negatively affected, and the increase in cell proliferation and inflammation may lead to loss of organization in the parasympathectomized lacrimal gland.

CATEGORY: BIOCHEMISTRY & MOLECULAR BIOLOGY; CELL BIOLOGY; PHYSIOLOGY
SUPPLEMENTARY TERM: muscarinic; dry **eye**
SUPPL. TERM PLUS: PAROTID ACINAR-CELLS; **SURFACTANT PROTEIN -D**; HUMAN **TEAR**-FLUID; SKELETAL-MUSCLE; GENE-EXPRESSION; ANNEXIN-I; PHYSIOLOGICAL RESPONSIVENESS; ADRENOCORTICOTROPIC HORMONE; ENDOPLASMIC-RETICULUM; SENSORY DENERVATION

REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	ARN PG (RPG)	Referenced Work (RWK)
ABRAHAMSON M	1987	216	229	FEBS LETT
ADHAM N	1987	18	517	GEN PHARMACOL
ARUR S	2003	4	587	DEV CELL
AZUMA T	2002	81	171	IMMUNOL LETT

BASS J	2000	97	11905	P NATL ACAD SCI USA
BISGAARD H C	2002	161	1187	AM J PATHOL
BITTO E	2000	39	13469	BIOCHEMISTRY-US
BRAUNEWELL K H	1999	295	1	CELL TISSUE RES
BROSTROM C O	1990	52	577	ANNU REV PHYSIOL
CHENG H	1996	244	327	ANAT REC
CHEN W B	1997	91	789	CELL
CREAGH E M	2003	193	10	IMMUNOL REV
CRIPPS M M	1987	45	673	EXP EYE RES
DEDHAR S	1994	367	480	NATURE
DEHOSTOS E L	1999	9	345	TRENDS CELL BIOL
DELBONO O	2003	2	21	AGING CELL
DICKINSON D P	2002	21	47	DNA CELL BIOL
DIDICHENKO S A	2000	485	147	FEBS LETT
EDMAN J C	1985	317	267	NATURE
EDWARDS A V	2002	100	50	AUTON NEUROSCI-BASIC
EKSTROM J	2001	46	1151	ARCH ORAL BIOL
EVANS R L	1997	499	351	J PHYSIOL-LONDON
EVANS R L	2000	275	26720	J BIOL CHEM
FLUCK M	2003	146	159	REV PHYSIOL BIOCH P
FOX R I	1994	350	609	ADV EXP MED BIOL
FREEDMAN R B	2002	3	136	EMBO REP
FREY B M	1999	13	2235	FASEB J
FUJIHARA T	2001	42	S258	INVEST OPHTH VIS S S
GIRARD L R	1993	268	26592	J BIOL CHEM
HELGREN M E	1994	76	493	CELL
HINCKE M T	1992	282	877	BIOCHEM J 3
JAHN R	1982	126	623	EUR J BIOCHEM
KISELYOV K	2002	506	175	ADV EXP MED BIOL
KRANN M V D	1998	139	2348	ENDOCRINOLOGY
LI X J	1995	270	17674	J BIOL CHEM
LIU N G	1994	269	28635	J BIOL CHEM
LIU Y Q	1995	306	637	BIOCHEM J
MA Z M	1997	272	11118	J BIOL CHEM
MADSEN J	2003	33	2327	EUR J IMMUNOL
MATSUI M	2000	97	9579	P NATL ACAD SCI USA
MENERAY M A	1998	17	99	CORNEA
MENERAY M A	1994	35	4144	INVEST OPHTH VIS SCI
MEUNIER L	2002	13	4456	MOL BIOL CELL
MICHALAK M	1999	344	281	BIOCHEM J 2
MIRCHEFF A K	1996	4	1	OCUL IMMUNOL INFLAMM
MIRCHEFF A K	1994	35	3943	INVEST OPHTH VIS SCI
MOSER M	2002	22	1438	MOL CELL BIOL
NAKAYAMA K	1997	327	625	BIOCHEM J 3
NGUYEN D	2002			ASS RES VIS OPHT MAY
NGUYEN D H	2002	506	65	ADV EXP MED BIOL
PAUSE A	1994	371	762	NATURE
RAYNAL P	1994	1197	63	BIOCHIM BIOPHYS ACTA
REDL B	2000	1482	241	BBA-PROTEIN STRUCT M
RICHARDS S M	2002	506	121	ADV EXP MED BIOL
ROSSI J	1999	22	243	NEURON
SATHE S	1998	17	348	CURR EYE RES
SCHIMMELPFENNIG B	1982	218	287	GRAEFES ARCH CLIN EX
SCHULZ B L	2002	366	511	BIOCHEM J 2
SCOTT B L	1959	104	115	AM J ANAT
SHAW P A	2001	70	301	LIFE SCI
SIVAKUMAR S	1998	252	485	ANAT REC
SPRONG H	2003	14	3482	MOL BIOL CELL
STAHLMAN M T	2002	50	651	J HISTOCHEM CYTOCHEM
STERN M E	1998	17	584	CORNEA

STERN M E	1998	438	643	ADV EXP MED BIOL
TAKITO J	1999	277	F277	AM J PHYSIOL-RENAL
TALAMO B R	1979	24	1573	LIFE SCI
TAO Y	1999	277	C994	AM J PHYSIOL-CELL PH
TENNETI L	1998	273	26799	J BIOL CHEM
TOMA L	1996	271	3897	J BIOL CHEM
TOSHIDA H	2002	506	225	ADV EXP MED BIOL
UCHIDA K	2003	88	394	J CELL BIOCHEM
WALCOTT B	2002	506	191	ADV EXP MED BIOL
WOOD R L	1999	69	213	EXP EYE RES
YOSHINO K	1996	15	617	CORNEA
ZOUKHRI D	1995	268	C713	AM J PHYSIOL-CELL PH

L148 ANSWER 45 OF 45 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:754187 SCISEARCH Full-text<<LOGINID::20061004>>

THE GENUINE ARTICLE: 593DE

TITLE: Identification of two highly sialylated human **tear** -fluid DMBT1 isoforms: the major high-molecular-mass glycoproteins in human **tears**

AUTHOR: Schulz B L; Oxley D; Packer N H; Karlsson N G (Reprint)

CORPORATE SOURCE: Proteome Syst Ltd, Locked Bag 2073, Sydney, NSW 1670, Australia (Reprint); Proteome Syst Ltd, Sydney, NSW 1670, Australia

COUNTRY OF AUTHOR: Australia

SOURCE: BIOCHEMICAL JOURNAL, (1 SEP 2002) Vol. 366, Part 2, pp. 511-520.
ISSN: 0264-6021.

PUBLISHER: PORTLAND PRESS, 59 PORTLAND PLACE, LONDON W1N 3AJ, ENGLAND

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 30

ENTRY DATE: Entered STN: 4 Oct 2002
Last Updated on STN: 4 Oct 2002

ABSTRACT:

Human open **eye tear** fluid was separated by low-percentage SDS/PAGE to detect high-molecular-mass protein components. Two bands were found with apparent molecular masses of 330 and 270 kDa respectively. By peptide-mass fingerprinting after tryptic digestion, the proteins were found to be isoforms of the DMBT1 gene product, with over 30% of the predicted protein covered by the tryptic peptides. By using gradient SDS/agarose/polyacrylamide composite gel electrophoresis and staining for glycosylation, it was shown that the two isoforms were the major high-molecular-mass glycoproteins of > 200 kDa in human **tear** fluid. Western blotting showed that the proteins expressed sialyl-Le(a). After the release of oligosaccharides by reductive beta-elimination from protein blotted on to PVDF membrane, it was revealed by liquid chromatography-MS that the O-linked oligosaccharides were comprised mainly of highly sialylated oligosaccharides with up to 16 monosaccharide units. A majority of the oligosaccharides could be described by the formula dHex(0-->2)NeuAc(1-->x) He(x)HexNAc(x)(-ol), x=1-6, where Hex stands for hexose, dHex for deoxyhexose, HexNAc for N-acetylhexosamine and NeuAc for N-acetylneuraminate. The number of sialic acids in the formula is less than 5. Interpretation of collision-induced fragmentation tandem MS confirmed the presence of sialic acid and suggested the presence of previously undescribed structures carrying the sialyl-Le(a) epitopes. Small amounts of neutral and sulphated species were also present. This is the first time that O-linked oligosaccharides have been detected and described from protein variant of the DMBT1 gene.

CATEGORY: BIOCHEMISTRY & MOLECULAR BIOLOGY

SUPPLEMENTARY TERM: eye; glycosylation; mucin; O-linked oligosaccharide; sialyl-Le(a); tear film

SUPPL. TERM PLUS: **SURFACTANT PROTEIN-D**; HUMAN CONJUNCTIVA; SRCR SUPERFAMILY; HUMAN CORNEAL; MUCIN; RECEPTOR; GP-340; MEMBER; ORIGIN; MUCUS

REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	ARN PG (RPG)	Referenced Work (RWK)
BERRY M	1996	37	2559	INVEST OPHTH VIS SCI
CAPON C	1992	267	19248	J BIOL CHEM
DOMON B	1988	5	397	GLYCOCONJUGATE J
ELLINGHAM R B	1999	9	1181	GLYCOBIOLOGY
FLEISZIG S M J	1994	62	1799	INFECT IMMUN
GARCHER C	1994	35	1184	INVEST OPHTH VIS SCI
GIPSON I K	1992	33	218	INVEST OPHTH VIS SCI
HELLMAN U	1995	224	451	ANAL BIOCHEM
HERBERT B	2001	22	2046	ELECTROPHORESIS
HIKITA C	2000	151	1235	J CELL BIOL
HOLMSKOV U	1999	96	10794	P NATL ACAD SCI USA
HOLMSKOV U	1997	272	13743	J BIOL CHEM
INATOMI T	1995	36	1818	INVEST OPHTH VIS SCI
JUMBLATT M M	1999	40	43	INVEST OPHTH VIS SCI
KHYSEANDERSON J	1984	10	203	J BIOCHEM BIOPH METH
MAWHINNEY T P	1992	235	179	CARBOHYD RES
MCKENZIE R W	2000	41	703	INVEST OPHTH VIS SCI
MOLLENHAUER J	1997	17	32	NAT GENET
NILSSON L A	1987	1	49	J DERMATOL SURG ONC
NUNES D P	1995	310	41	BIOCHEM J
PFLUGFELDER S C	2000	41	1316	INVEST OPHTH VIS SCI
PRAKOBPHOL A	2000	275	39860	J BIOL CHEM
PRAKOBPHOL A	1998	37	4916	BIOCHEMISTRY-US
PRYDAL J I	1993	7	472	EYE
REDL B	2000	1482	241	BBA-PROTEIN STRUCT M
RESNICK D	1994	19	5	TRENDS BIOCHEM SCI
SACK R A	1998	438	235	ADV EXP MED BIOL
SACK R A	1997	16	577	CURR EYE RES
TINO M J	1999	20	759	AM J RESP CELL MOL
WATANABE H	1995	36	337	INVEST OPHTH VIS SCI

FILE 'HOME' ENTERED AT 14:59:55 ON 04 OCT 2006

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**********SEARCH HISTORY**********
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     (FILE 'HOME' ENTERED AT 13:36:06 ON 04 OCT 2006)

     FILE 'CAPLUS' ENTERED AT 13:36:26 ON 04 OCT 2006
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                D SCAN
L2           33 SEA ABB=ON  FLEISZIG S?/AU
L3         5041 SEA ABB=ON  EVANS D?/AU
L4          246 SEA ABB=ON  SACK R?/AU
L5            2 SEA ABB=ON  L2 AND L3 AND L4
L6         9103 SEA ABB=ON  COLLECTIN#/OBI
L7         4798 SEA ABB=ON  SURFACTANT/OBI(L)PROTEIN#/OBI
                E EYE, DISEASE+NT/CT
                E EYE, DISEASE+ALL/CT
L8        26855 SEA ABB=ON  EYE#/OBI(L)(DISEASE#/OBI OR DISORDER#/OBI)
L9         8564 SEA ABB=ON  OCULAR/OBI
L10       10251 SEA ABB=ON  OPHTHALM?/OBI
L11        4763 SEA ABB=ON  CONTACT LENS?/OBI
L12           3 SEA ABB=ON  (L2 OR L3 OR L4) AND (L6 OR L7)
L13         635 SEA ABB=ON  SP D/OBI
L14          12 SEA ABB=ON  L6 AND (L8 OR L9 OR L10 OR L11)
L15         225 SEA ABB=ON  L6 NOT COLLECTING/OBI
L16           5 SEA ABB=ON  L15 AND (L8 OR L9 OR L10 OR L11)
                D SCAN TI
                D SCAN
L17         171 SEA ABB=ON  ANTIMICROBIAL#/OBI(L)LENS?/OBI
L18           1 SEA ABB=ON  L17 AND L15
L19           1 SEA ABB=ON  (L7 OR L13) AND L17
L20          21 SEA ABB=ON  (L7 OR L13) AND (L8 OR L9 OR L10 OR L11)
L21          19 SEA ABB=ON  L20 NOT (L1 OR L5 OR L12 OR L16 OR L18 OR L19)
                D SCAN L1
                E SURFACTANT PROTEINS/CT
                E E4+ALL
L22           6 SEA ABB=ON  L13 AND (L8 OR L9 OR L10 OR L11)
                D SCAN TI
L23           1 SEA ABB=ON  TEAR/TI AND L22
                D SCAN
L24       76046 SEA ABB=ON  EYE/CT
                E TEAR/CT
                E E6+ALL
L25        2234 SEA ABB=ON  TEAR#/CW
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L26           0 SEA ABB=ON  (L7 OR L13) AND FIL STNGUIDE/OBI

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L29         327 SEA ABB=ON  (L7 OR L13)(L)(THU OR PAC OR PKT OR DMA)/RL
L30           9 SEA ABB=ON  L29 AND (L8 OR L9 OR L10 OR L11 OR L24 OR L25)
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L31         419 SEA ABB=ON  SURFACTANT FREE/OBI
L32           8 SEA ABB=ON  L29 AND (L8 OR L9 OR L10 OR L11 OR L24 OR L25) NOT
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                D QUE L18

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L33           48 SEA ABB=ON  FLEISZIG S?/AU
L34         4088 SEA ABB=ON  EVANS D?/AU
L35          413 SEA ABB=ON  SACK R?/AU
L36         2293 SEA ABB=ON  COLLECTINS+NT/CT
L37       320667 SEA ABB=ON  EYE DISEASES+NT/CT
L38        13816 SEA ABB=ON  KERATITIS+NT/CT
L39         9822 SEA ABB=ON  DRY EYE SYNDROMES+NT/CT
L40         8529 SEA ABB=ON  CONTACT LENSES+NT/CT
L41         4849 SEA ABB=ON  TEARS/CT
L42            1 SEA ABB=ON  (L33 AND L34 AND L35) OR ((L33 OR L34 OR L35) AND
                  L36)
                D TRIAL
L43       221621 SEA ABB=ON  EYE+NT/CT
L44        22450 SEA ABB=ON  PSEUDOMONAS AERUGINOSA/CT
L45            1 SEA ABB=ON  (L43 OR L37) AND L44 AND L36
L46           12 SEA ABB=ON  L36 AND L37
                D TRIAL 1-12
L47          202 SEA ABB=ON  L36(L)(BL OR BI)/CT
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L49         1219 SEA ABB=ON  ACANTHAMOEBA/CT
L50            1 SEA ABB=ON  L49 AND L36 AND L37
L51            0 SEA ABB=ON  L36 AND L40
L52            1 SEA ABB=ON  L36 AND L41
L53            1 SEA ABB=ON  L36 AND (L41 OR L40)
L54           89 SEA ABB=ON  L36(L)(AD OR PD OR PK OR TU)/CT
L55            4 SEA ABB=ON  L54 AND (L43 OR L44 OR L37)
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L57         3264 SEA ABB=ON  EVANS D?/AU
L58          299 SEA ABB=ON  SACK R?/AU
                E COLLECTIN/CT
                E E3+ALL
L59          221 SEA ABB=ON  COLLECTIN/CT
                E PULMONARY SURFACTANT-ASS/CT
                E E7+ALL
                E E2+ALL
L60          535 SEA ABB=ON  SURFACTANT PROTEIN D/CT
                E PULMONARY SURFACTANT-ASSOCIATED PROTEINS+ALL/CT
                E E2+ALL
L61          375 SEA ABB=ON  SURFACTANT ASSOCIATED PROTEIN/CT
                E SURFACTANT ASSOCIATED PROTEIN+NT/CT
                E EYE DISEASE+ALL/CT
                E EYE INFECTIONS+ALL/CT
                E E2+ALL
L62       302195 SEA ABB=ON  EYE DISEASE+NT/CT
                E CONTACT LENS/CT
                E E3+ALL
L63         5975 SEA ABB=ON  CONTACT LENS/CT
                E TEARS+ALL/CT
                E E2+ALL
L64         1416 SEA ABB=ON  LACRIMAL FLUID/CT
                E ARTIFICIAL TEARS/CT
L65          900 SEA ABB=ON  ARTIFICIAL TEAR/CT
L66            1 SEA ABB=ON  (L56 AND L57 AND L58) OR ((L56 OR L57 OR L58) AND
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                (L59 OR L60 OR L61))
                D TRIAL
L67          932 SEA ABB=ON  EYE PROTECTION/CT
L68       132501 SEA ABB=ON  EYE+NT/CT
L69        30005 SEA ABB=ON  PSEUDOMONAS AERUGINOSA/CT
L70         4680 SEA ABB=ON  INFECTION RESISTANCE/CT
L71            7 SEA ABB=ON  (L59 OR L60 OR L61) AND L62
                D TRIAL 1-7
L72          665 SEA ABB=ON  (L59 OR L60 OR L61)(L)EC/CT
L73            1 SEA ABB=ON  (L59 OR L60 OR L61) AND L62 NOT L72
L74            0 SEA ABB=ON  (L59 OR L60 OR L61) AND L63
L75            0 SEA ABB=ON  (L59 OR L60 OR L61) AND (L64 OR L65)
L76            0 SEA ABB=ON  (L59 OR L60 OR L61) AND (L63 OR L64 OR L65)
L77            1 SEA ABB=ON  (L59 OR L60 OR L61) AND L67
L78           47 SEA ABB=ON  (L59 OR L60 OR L61) AND (L62 OR (L68 OR L69 OR
                L70))
L79           12 SEA ABB=ON  (L59 OR L60 OR L61) AND (L62 OR (L68 OR L69 OR
                L70)) NOT L72
                D TRIAL 1-12
L80           50 SEA ABB=ON  (L59 OR L60 OR L61)(L)(AD OR DO OR DV OR CT OR DT
                OR PD OR PK)/CT
L81            3 SEA ABB=ON  L80 AND (L62 OR (L68 OR L69 OR L70)) NOT L72

     FILE 'WPIX' ENTERED AT 14:29:58 ON 04 OCT 2006
L82            3 SEA ABB=ON  FLEISZIG S?/AU
L83          848 SEA ABB=ON  EVANS D?/AU
L84           32 SEA ABB=ON  SACK R?/AU
L85           51 SEA ABB=ON  COLLECTIN/BI,ABEX OR COLLECTINS/BI,ABEX OR
                COLLAGEN LIKE LECTIN#/BI,ABEX
L86          318 SEA ABB=ON  SPD/BI,ABEX OR (SP/BI,ABEX OR SURFACTANT/BI,ABEX(1W
                )PROTEIN/BI,ABEX)(W)D/BI,ABEX
L87        93281 SEA ABB=ON  EYE?/BI,ABEX
L88        11427 SEA ABB=ON  OCULAR?/BI,ABEX
L89        23904 SEA ABB=ON  OPHTHALM?/BI,ABEX
L90         8316 SEA ABB=ON  CONTACT LENS?/BI,ABEX
L91         1028 SEA ABB=ON  KERATITIS/BI,ABEX
L92            1 SEA ABB=ON  (L82 AND L83 AND L84) OR ((L82 OR L83 OR L84) AND
                (L85 OR L86))
                D TRIAL
                E B04-N02+ALL/MC
                E B14-N03+ALL/MC
                E D09-C01A+ALL/MC
L93         4610 SEA ABB=ON  D09-C01A/MC
L94        17613 SEA ABB=ON  B14-N03/MC OR C14-N03/MC
                E COLLECTIN/CN
L95           22 SEA ABB=ON  (L85 OR L86) AND (L87 OR L88 OR L89 OR L90 OR L91
                OR L93 OR L94)
L96            6 SEA ABB=ON  L85 AND (L87 OR L88 OR L89 OR L90 OR L91 OR L93 OR
                L94)
L97           17 SEA ABB=ON  L86 AND (L87 OR L88 OR L89 OR L90 OR L91 OR L93 OR
                L94)
L98           12 SEA ABB=ON  L97 AND B/DC
                D TRIAL 1-12
L99           11 SEA ABB=ON  L98 NOT (L92 OR L96)
                D KWIC 1-3
L100          66 SEA ABB=ON  (SP/BI,ABEX OR SURFACTANT/BI,ABEX(1W)PROTEIN/BI,ABE
                X)(W)D/BI,ABEX
L101           3 SEA ABB=ON  L100 AND (L87 OR L88 OR L89 OR L90 OR L91 OR L93
                OR L94)
L102       30746 SEA ABB=ON  TEAR#/BI,ABEX
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L103          1 SEA ABB=ON  L102 AND (L100 OR L85)
L104        307 SEA ABB=ON  SURFACTANT/BI,ABEX(1W)PROTEIN#/BI,ABEX
L105         11 SEA ABB=ON  L104 AND (L87 OR L88 OR L89 OR L90 OR L91 OR L93
                  OR L94 OR L102)
L106          7 SEA ABB=ON  L105 NOT (L92 OR L96 OR L101 OR L103)
                  D TRIAL 1-7
L107          1 SEA ABB=ON  L106 AND BEADLET?/TI
                  D KWIC

     FILE 'DRUGU' ENTERED AT 14:39:48 ON 04 OCT 2006
L108          0 SEA ABB=ON  FLEISZIG S?/AU
L109        397 SEA ABB=ON  EVANS D?/AU
L110         31 SEA ABB=ON  SACK R?/AU
L111          0 SEA ABB=ON  L109 AND L110
                  E EYE/CT
L112       7295 SEA ABB=ON  EYE+NT/CT
L113      14910 SEA ABB=ON  EYE -DISEASE/CT OR EYE-DISEASE+NT/CT
                  E E28+ALL
                  E INFECTION/CT
L114     129709 SEA ABB=ON  INFECTION#
                  E COLLECTIN/CT

     FILE 'STNGUIDE' ENTERED AT 14:42:17 ON 04 OCT 2006

     FILE 'DRUGU' ENTERED AT 14:45:51 ON 04 OCT 2006
L115          7 SEA ABB=ON  COLLECTIN OR COLLECTINS OR COLLAGEN LIKE LECTIN#
L116         94 SEA ABB=ON  (SP OR SURFACTANT(1W)PROTEIN)(W)D
L117         68 SEA ABB=ON  (L109 OR L110) AND (L112 OR L113 OR L114 OR L115
                  OR L116)
L118          0 SEA ABB=ON  (L109 OR L110) AND (L115 OR L116)
L119        983 SEA ABB=ON  TEAR#
L120       1525 SEA ABB=ON  LENS OR LENSES
L121          0 SEA ABB=ON  (L115 OR L116) AND (L119 OR L120 OR L112 OR L113)

     FILE 'STNGUIDE' ENTERED AT 14:47:57 ON 04 OCT 2006

     FILE 'JICST-EPLUS, CABA, BIOSIS, LIFESCI, CONFSCI, DISSABS, SCISEARCH'
     ENTERED AT 14:50:34 ON 04 OCT 2006
L122        254 SEA ABB=ON  FLEISZIG S?/AU
L123      16140 SEA ABB=ON  EVANS D?/AU
L124       1188 SEA ABB=ON  SACK R?/AU
L125     519542 SEA ABB=ON  EYE?
L126     183964 SEA ABB=ON  OCULAR? OR INTRAOCULAR?
L127     149128 SEA ABB=ON  OPHTHALM?
L128     150518 SEA ABB=ON  LENS OR LENSES
L129      16862 SEA ABB=ON  KERATITIS
L130      38386 SEA ABB=ON  TEAR#
L131          0 SEA ABB=ON  CONJUCTIVITIS SICCA
L132       1648 SEA ABB=ON  XEROPHTHALM?
L133      17416 SEA ABB=ON  SJOGREN?
L134       1201 SEA ABB=ON  COLLECTIN OR COLLECTINS OR COLLAGEN LIKE LECTIN#
L135       3121 SEA ABB=ON  (SP OR SURFACTANT(1W) PROTEIN)(W) D
L136        177 SEA ABB=ON  CONJUNCTIVITIS SICCA
L137          8 SEA ABB=ON  (L122 AND L123 AND L124) OR ((L122 OR L123 OR
                  L124) AND (L134 OR L135))
L138         34 SEA ABB=ON  (L134 OR L135) AND (L125 OR L126 OR L127 OR L128
                  OR L129 OR L130 OR L132 OR L133 OR L136)
L139         22 DUP REM L138 (12 DUPLICATES REMOVED)
                       ANSWERS '1-2' FROM FILE JICST-EPLUS
                       ANSWER '3' FROM FILE CABA
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                    ANSWERS '4-17' FROM FILE BIOSIS
                    ANSWERS '18-19' FROM FILE DISSABS
                    ANSWERS '20-22' FROM FILE SCISEARCH
L140          64 SEA ABB=ON  L134(W) RECEPTOR#
L141          31 SEA ABB=ON  L138 NOT L140

     FILE 'STNGUIDE' ENTERED AT 14:54:06 ON 04 OCT 2006

     FILE 'JICST-EPLUS, CABA, BIOSIS, LIFESCI, CONFSCI, DISSABS, SCISEARCH'
     ENTERED AT 14:55:25 ON 04 OCT 2006
               D QUE L137

     FILE 'DRUGU' ENTERED AT 14:55:26 ON 04 OCT 2006
               D QUE L108
               D QUE L118
               D QUE L111

     FILE 'MEDLINE' ENTERED AT 14:55:28 ON 04 OCT 2006
               D QUE L42

     FILE 'EMBASE' ENTERED AT 14:55:28 ON 04 OCT 2006
               D QUE L66

     FILE 'WPIX' ENTERED AT 14:55:28 ON 04 OCT 2006
               D QUE L92

     FILE 'CAPLUS' ENTERED AT 14:55:29 ON 04 OCT 2006
               D QUE L1
               D QUE L12
               D QUE L5
L142           3 SEA ABB=ON  (L1 OR L5 OR L12)

     FILE 'MEDLINE, CAPLUS, WPIX, EMBASE, BIOSIS, LIFESCI, SCISEARCH' ENTERED
     AT 14:55:52 ON 04 OCT 2006
L143           8 DUP REM L42 L142 L92 L66 L137 (6 DUPLICATES REMOVED)
                    ANSWER '1' FROM FILE MEDLINE
                    ANSWERS '2-3' FROM FILE CAPLUS
                    ANSWER '4' FROM FILE WPIX
                    ANSWERS '5-7' FROM FILE BIOSIS
                    ANSWER '8' FROM FILE SCISEARCH
               D IBIB ED ABS 1-8

     FILE 'STNGUIDE' ENTERED AT 14:56:12 ON 04 OCT 2006

     FILE 'JICST-EPLUS, CABA, BIOSIS, LIFESCI, CONFSCI, DISSABS, SCISEARCH'
     ENTERED AT 14:58:32 ON 04 OCT 2006
               D QUE L141

     FILE 'DRUGU' ENTERED AT 14:58:33 ON 04 OCT 2006
               D QUE L121

     FILE 'CAPLUS' ENTERED AT 14:58:35 ON 04 OCT 2006
               D QUE L16
               D QUE L18
               D QUE L19
               D QUE L32
L144          11 SEA ABB=ON  (L16 OR L18 OR L19 OR L32) NOT L142

     FILE 'EMBASE' ENTERED AT 14:58:37 ON 04 OCT 2006
               D QUE L73
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                D QUE L76
                D QUE L77
                D QUE L81
L145          4 SEA ABB=ON  (L73 OR L77 OR L81) NOT L66

     FILE 'WPIX' ENTERED AT 14:58:40 ON 04 OCT 2006
                D QUE L96
                D QUE L101
                D QUE L103
L146          7 SEA ABB=ON  (L96 OR L101 OR L103) NOT L92

     FILE 'MEDLINE' ENTERED AT 14:58:43 ON 04 OCT 2006
                D QUE L45
                D QUE L48
                D QUE L50
                D QUE L52
                D QUE L55
L147          9 SEA ABB=ON  (L45 OR L48 OR L50 OR L52 OR L55) NOT L42

     FILE 'STNGUIDE' ENTERED AT 14:58:51 ON 04 OCT 2006

     FILE 'MEDLINE, CAPLUS, WPIX, EMBASE, JICST-EPLUS, CABA, BIOSIS, LIFESCI,
     DISSABS, SCISEARCH' ENTERED AT 14:59:10 ON 04 OCT 2006
L148         45 DUP REM L147 L144 L146 L145 L141 (17 DUPLICATES REMOVED)
                       ANSWERS '1-9' FROM FILE MEDLINE
                       ANSWERS '10-20' FROM FILE CAPLUS
                       ANSWERS '21-23' FROM FILE WPIX
                       ANSWERS '24-26' FROM FILE EMBASE
                       ANSWERS '27-28' FROM FILE JICST-EPLUS
                       ANSWER '29' FROM FILE CABA
                       ANSWERS '30-40' FROM FILE BIOSIS
                       ANSWERS '41-42' FROM FILE DISSABS
                       ANSWERS '43-45' FROM FILE SCISEARCH
                D IALL 1-9
                D IBIB ED ABS HITIND 10-20
                D IALL ABEQ TECH 21-23
                D IALL 24-45

     FILE 'HOME' ENTERED AT 14:59:55 ON 04 OCT 2006

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